# IMPACT OF ALCOHOL ON AUDITORY THRESHOLDS

**Dissertation submitted to**


**THE TAMILNADU DR. M.G.R MEDICAL UNIVERSITY**

**CHENNAI – 600032.**

**In partial fulfillment of the requirement for the degree of**

**Doctor of Medicine in Physiology (Branch V)**

**M.D. (PHYSIOLOGY)**

**APRIL 2015**

**DEPARTMENT OF PHYSIOLOGY**

**COIMBATORE MEDICAL COLLEGE**

**COIMBATORE – 14.**

# CERTIFICATE

This dissertation entitled **"IMPACT OF ALCOHOL ON AUDITORY THRESHOLDS"** is submitted to The Tamil Nadu Dr.M.G.R. Medical University, Chennai, in partial fulfillment of regulations for the award of M.D. Degree in Physiology in the examinations to be held during April 2015.

This dissertation is a record of fresh work done by the candidate **Dr. N. SIVAKUMAR,** during the course of the study (2012-2015).

This work was carried out by the candidate himself under my supervision.

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## DECLARATION

I **Dr. N. SIVAKUMAR** solemnly declare that the dissertation entitled **"IMPACT OF ALCOHOL ON AUDITORY THRESHOLDS"** was done by me at Coimbatore Medical College, during the period from August 2013 to June 2014 under the guidance and supervision of **Dr. P. Murugesan. M.D.,** Associate Professor, Department of Physiology, Coimbatore Medical College, Coimbatore.

This dissertation is submitted to The Tamilnadu Dr. M.G.R. Medical University towards the partial fulfillment of the requirement for the award of M.D. Degree (Branch - V) in Physiology. I have not submitted this dissertation on any previous occasion to any University for the award of any degree.

**Place:**

**Date:** **Dr. N. Sivakumar.**

# Coimbatore Medical College

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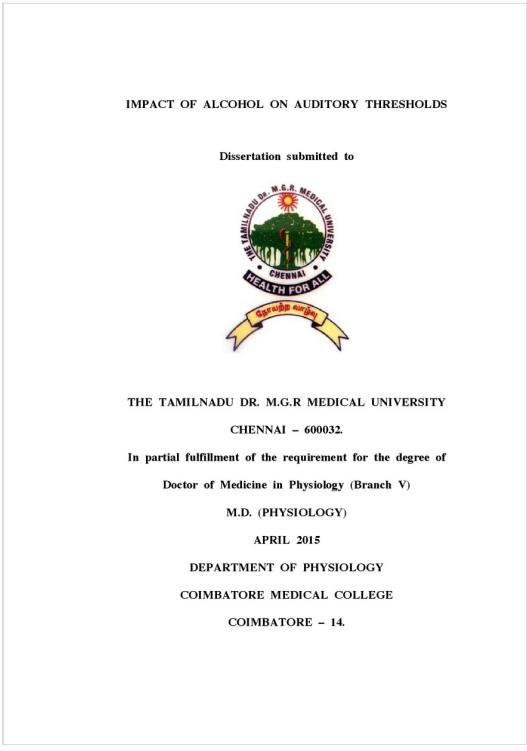

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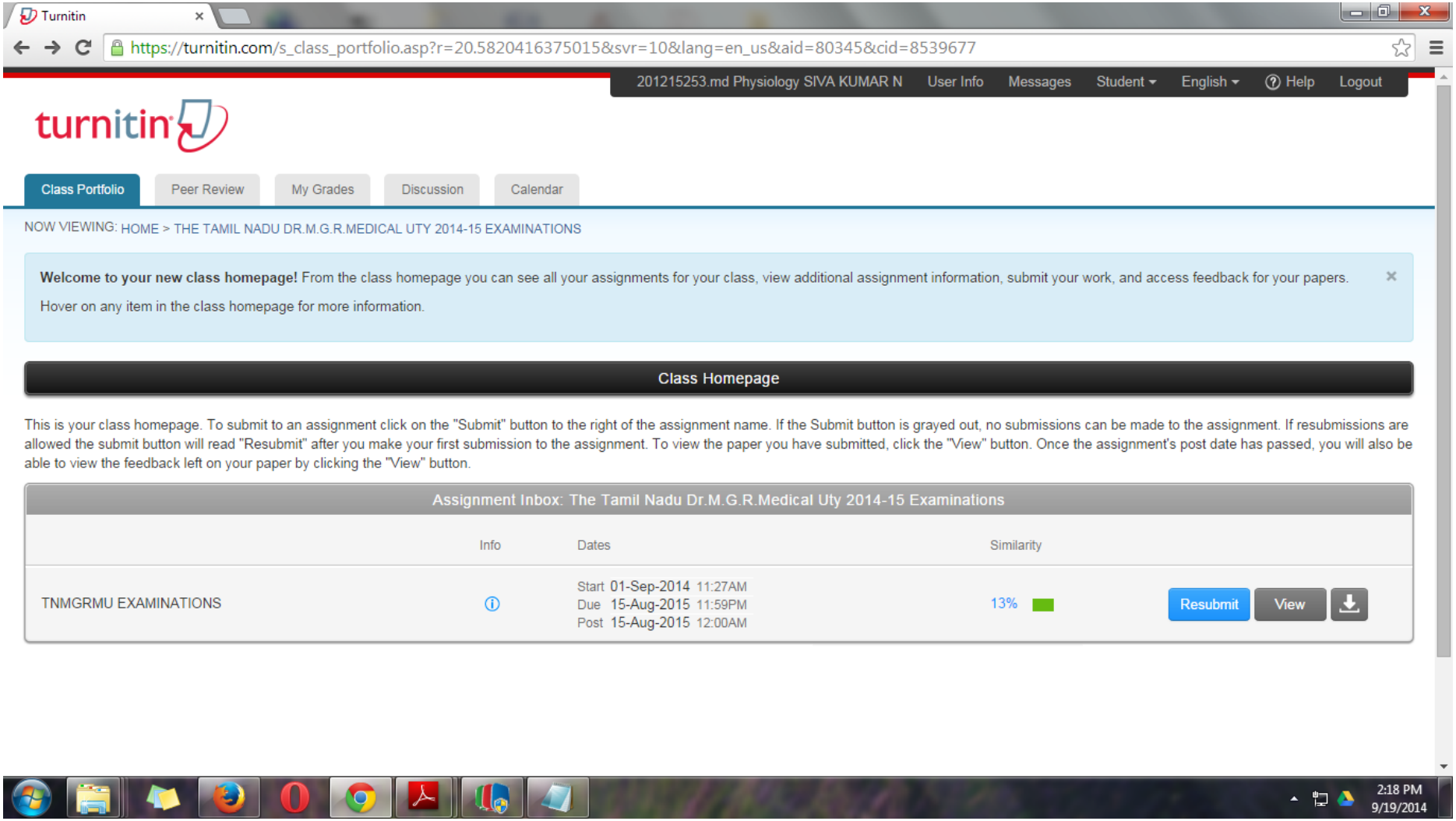
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## ABBREVIATIONS USED IN THE STUDY

- ASHA : American Speech Language Hearing Association
- AUDIT : The Alcohol Use Disorders Identification Test
- DB : Decibel
- DPOAE : Distortion Product Oto Acoustic Emissions
- $GABA_A$ : Gamma-Amino Butyric Acid 'A' Receptor
- HZ : Hertz
- NMDA : N-Methyl D-Aspartate Receptor
- OAE : Oto Acoustic Emission
- OHCS : Outer Hair Cells
- PTA : Puretone Audiometry
- SPL : Sound Pressure Level
- SNHL : Sensori Neural Hearing Loss
- TEOAE : Transient Evoked Oto Acoustic Emission
- WHO : World Health Organisation

# IMPACT OF ALCOHOL ON AUDITORY THRESHHOLD

Department of Physiology, Coimbatore Medical College, Coimbatore.

## ABSTRACT

**BACK ROUND**: Alcohol is one of the serious public health problems and has become a common tendency worldwide. In general, approximately 2 billion of the world's population consumes alcohol and 76.3 million people have alcoholic disorders. Alcohol affects almost all organs of the body and causes cirrhosis, peripheral neuropathy, hyper tension, myocardial infarction and death. Alcohol also causes hearing loss.

**AIM & OBJECTIVE:** The aim of the study is to analyze the hearing loss in alcoholic men and non alcoholic men by using Puretone audiometry and smart DPOAE.

**DESIGN:** Descriptive with purposive sampling.

**PARTICIPANTS:** A total of 134 subjects (25 to 55 years) were included in the study of which 67 alcoholic men were taken as study group and 67 healthy non- alcoholics as controls. The study subjects were selected by AUDIT questionnaire from the outpatient department of psychiatry, Coimbatore medical college hospital, Coimbatore. Alcoholic men who were diabetic, hypertensive, smoker, subjects using ototoxic drugs were identified and excluded from the study.

**METHODOLOGY:** After getting consent, the cases were selected after ruling out any infections or foreign body in the external auditory meatus by using otoscope. After clearly explaining the procedure, audiometric thresholds were recorded by using puretone audiometer and smart DPOAE.

**RESULTS:** Unpaired't' test and Yate's and Fisher's chi square tests were used to analyze the significance of difference between variables. 44 out of 67 alcoholics and only 5 non-smokers were affected with sensorineural hearing loss ($p<0.0001$). There is significant increase in auditory thresholds in alcoholics ($p<0.0001$).

**CONCLUSION:** The results of this study suggest that, alcoholics were affected by high frequency (above 4 KHz) sensorineural hearing loss. The hearing loss is directly related to the duration, amount, type, occupation and age of alcoholics. Hearing loss may be due to the effect of inhibition of transmission via $GABA_A$ type receptors and damage occurred in outer hair cells in the organ of corti.

**KEY WORDS**: Sensorineural hearing loss, Auditory thresholds, Puretone audiometry and Distortion product otoacoustic emission.

# INTRODUCTION

**"Addiction should never be treated as a crime. It has to be treated as a health problem".**

***Ralph Nader , American Activist in 1934***

Alcohol is one of the worldwide medical and social problems. For the past 30 to 40 years consumption of alcohol increases in both quantity and frequency of intake. Nowadays people start drinking at their early ages and may have a greater risk of alcoholic disorders in later life. Now people are viewing the alcohol as a sign of prestige and social status.

In worldwide, about 2 billion people are consuming alcoholic beverages and 76.3 million people have alcoholic disorders. Around 2.3 million people die because of alcohol related disorders. The deaths due to alcohol constitute around 3.7% of all deaths[1]. About 20 to 30% of adult male and 5% of adult females are consuming alcohol in India. About 28% of traffic injuries are due to alcohol, according to a study conducted recently in Bangaluru[1].

Worldwide males who consume alcohol are affected more than females. In males around 6 % of alcoholic deaths and 7.4% of DALYs (Disability Adjusted Life Years), whereas in females, 1.1% of alcoholic deaths and 1.4% of DALYs. Worldwide, alcohol is responsible for 20% of motor vehicle accident deaths (Global Health Risks by WHO) [2].

There are many factors that increase the prevalence of use of alcohol in the community. One of them is the easy availability of the alcohol. One of the study in the U.S. showed that, in eight grade itself 46% of adolescents tried alcohol and 77% adolescents have begun to drunk in high school, because of easy availability of alcohol[1.]. In Tamilnadu also, there is increase in alcohol related disorders, especially the deaths due to traffic accidents due to drunken drive[1]. World Health Organization classified alcohol dependence as a disease and have included it in the International Statistical Classification of diseases $10^{th}$ revision (ICD – 10). Similarly, American Psychiatric Association Diagnostic and Statistical Manual of Mental Disorders (DSM – IV) also agreed alcohol dependence as a disease[3].

Consumption of alcohol is associated with physical, psychological and social problems. It affects almost all systems of the body. In the nervous system, alcohol causes mild impaired judgment to severe shrinkage of brain[4]. Similarly, in the hepato biliary system, it causes of mild hepatitis to severe cirrhosis of the liver. Alcoholism leads Hypertension. It leads to 6 fold increase in the risk of myocardial infarction. It also causes heart failure. These effects are mainly due to the production of free radicals from the alcohol metabolism and it directly and indirectly affects all organs[4]. It also causes the impairment in cognitive functions.

According to the World Health Organization, more than 360 million people have moderate hearing impairment to profound deafness[5], in which one fourth of the hearing loss begins from childhood. The consequences of hearing impairment in childhood are delayed in language acquisition, inability to interpret speech sounds and reduction in communication skills.

It is very important to detect the Sensorineural hearing loss (SNHL) in an early stage, so that we can take appropriate measures and arrest its progress.

Some studies have even demonstrated the reversibility of hearing loss following early detection and intervention[6]. Sensorineural hearing loss is not reversible if detected in the later stage, and only the rehabilitation measures, such as hearing aids and other devices which helps the patient to communicate with others is possible.

Alcohol consumption causes tolerance to the sound. It is commonly encountered in developed countries, where parties at night with heavy drinking in a noisy environment is very common. This is the reason for the increase in the 'hearing aid clinic' in that country and the formation of a newer diagnosis like 'cocktail party deafness'[7].

Several investigators have reported that acute and chronic alcohol intake in larger doses, causes alterations in the auditory brain stem potentials and middle latency responses. Some studies have also showed that there is a temporary reduction in the otoacoustic emission amplitudes at high frequencies, when alcohol is consumed to toxic levels.

In the animal studies, congenitally, some animals had peripheral auditory dysfunctions with damage of hair cells. Some animals had central auditory disorders with the prolonged transmission of nerve potentials at the level of the brainstem. In Morizona et al showed in a guinea pig, that there was a reduction of cochlear microphonics, nerve action potentials and endocochlear potentials after the local application of ethanol in the inner ear[8]. Many studies showed that alcohol causes sensorineural hearing loss. The studies of Curgan SG, et al[9] and Lohle E, et al[10] proved that alcohol causes deficiency of vitamin $B_{12}$ and vitamin A and therefore causes the sensorineural hearing loss.

Sandra Beatriz et al[11] study showed that alcohol abuse in the long term causes damage to the outer hair cells. Very few studies like the one done by Nordhal et al have reported that there is no relation between alcohol abuse and hearing loss[12]. Sharon G. Curgan suggested that low and moderate level of alcohol consumption didn't cause hearing loss, but chronic alcohol abuse causes risk of hearing loss[9]. He also reported that the moderate level of alcohol consumption has cardio protective effect like anti thrombolytic activity and also prevents the disturbances in the cochlear blood flow[9].

There are many tests are available to analyze the auditory function like speech (voice) tests, standard tuning fork tests, pure tone audiometry, tympanometry, DPOAE (Distortion Product Oto Acoustic Emissions) recordings and BERA (Brain Stem Evoked Response Audiometry). The basic and gold standard procedure to evaluate the auditory threshold measurement is pure tone audiometry. It can be used to evaluate the results obtained by Questionnaire method[13]. It is most useful audiological measurement in which the subject can perceive a number of frequency sounds and able to record the findings. DPOAE recordings are useful in the subjects who are uncooperative and also useful in children.

While comparing hearing loss among alcoholics, different investigators have obtained different results with regard to the age, quantity and duration of consumption of alcohol. In India, there are very few studies regarding the hearing impairment associated with alcohol intake. The purpose of this study was to evaluate the relation between alcohol use and hearing loss in men.

# *AIMS & OBJECTIVES*

## AIM AND OBJECTIVES

1. To compare the hearing ability among alcoholics and normal individuals by using Pure tone audiometry and Distortion product otoacoustic emission test.
2. To identify the sensorineural hearing loss among the alcoholics.
3. To evaluate the correlation between the duration of alcoholism and hearing impairment.
4. To compare the audiometric findings in relation to the hearing loss and quantity (unit) of consumption of alcohol.
5. To assess the association between the type of alcohol and the hearing loss.
6. To evaluate the relation between occupation and hearing impairment due to alcohol intake.
7. To find the effect of age on hearing among alcoholics.

# *REVIEW OF LITERATURE*

# REVIEW OF LITERATURE

## The history regarding auditory physiology

In 5th century itself, the Greek physicians knew about the middle ear and tympanic membrane. The physicians described that the middle ear was the seat of hearing. The auditory nerve was described by Galen (131 - 201 AD) and he also described that it has come from the middle ear[14].

In 1543, Vesalius described about the ossicles, Malleus and Incus. In 1546, stapes, oval and round windows were described by Ingrassia. In 1561 cochlea, the labyrinth and the facial nerve canal were named by Fallopius. In 1564 Eustachius described the auditory tube. The sound pathway in the ear was traced by Coiter in 1566, based on the above discoveries.

In 1863, Duverneyin explained in detail about the sound pathway. In 1777, Meckel described about the presence of fluid in the labyrinth and he also described about the ice filling nature of the frozen temporal bone.

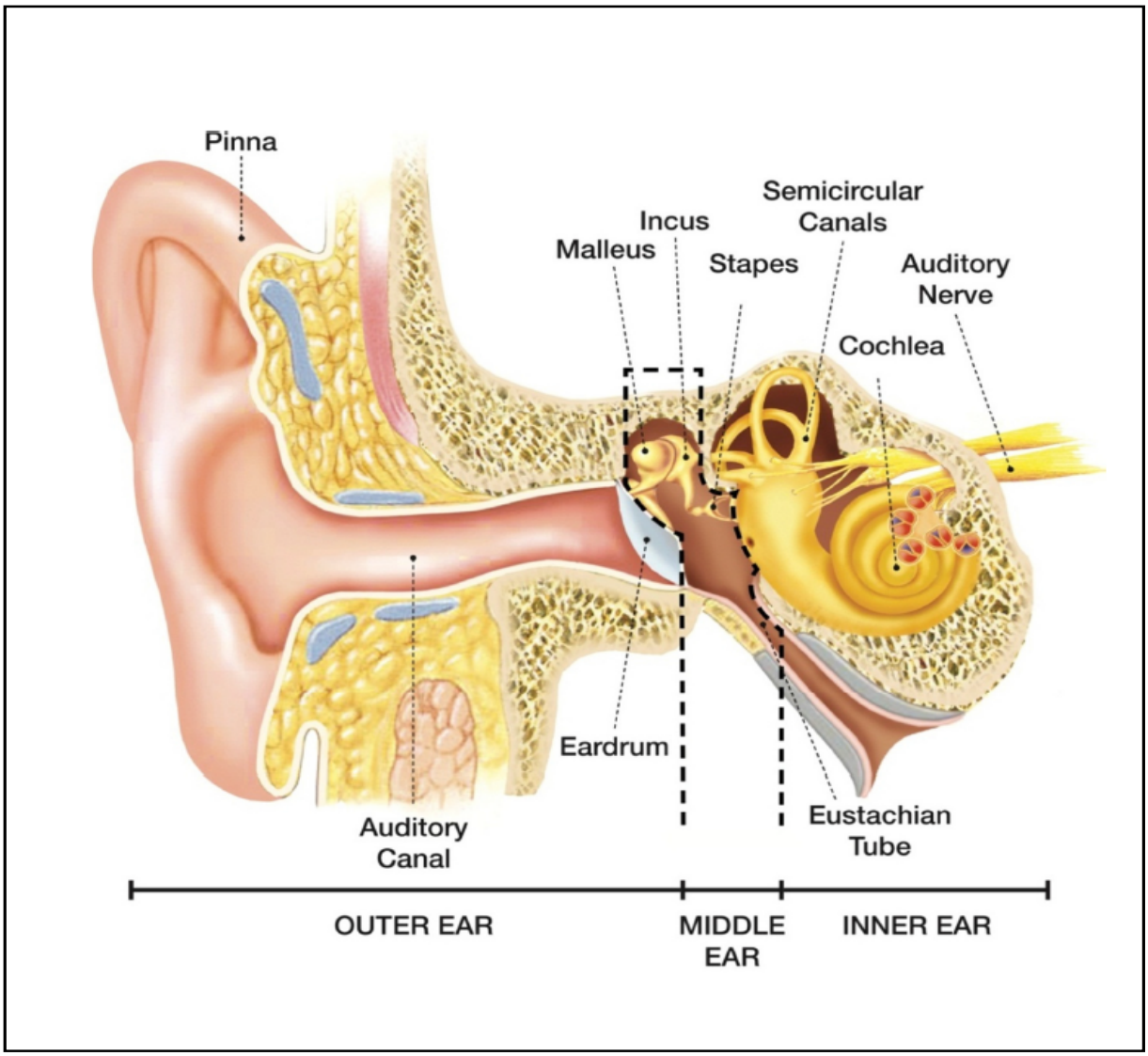
In 1851, Corti described in detail regarding the microscopic structures of the inner ear. The hair cells and their nerve connections are described by Retzius in 1892.

Helmholtz defined the principles of impedance matching and demonstrated the mechanism and functions of the middle ear in 1868. He also described the occurrence of the pressure transformation by three ways. They were, a lever action of the drum head, the lever actions by ossicular chain and a hydraulic action on the small footplate of stapes by the large tympanic membrane.

The middle ear mechanics were also contributed by George Von Bekesy, Josef Zwislocke, Ernst Glen Wever, William Peake, Merle Lawrence, Shyam Khanna, and Juergen Tonndorf.

In 1979, Kemp described that the cochlea receives sounds as well as it produce them. He also demonstrated that if a click sound was sent inside ear, cochlea emitted an echo for the click sound and he called it as Otoacoustic emissions[15]. In 1899, Carl E Seashore invented the audiometer. Auditory chart which included the tuning fork test, was designed by Arthur Hartmann in 1885.

# ANATOMY OF EAR



In human cadaveric cochleas, Von Bekesy demonstrated the cochlear partition. He also found that the basilar membrane has varying stiffness. Van Bekesy got the Nobel prize in Physiology/ Medicine in 1961 for the study of cochlear mechanics[16].

## Anatomy of the ear

### External ear

***Pinna*** (Auricle) has a thin plate of irregularly shaped yellow elastic cartilage and so it is flexible. Auricle is covered by perichondrium and skin. The external auditory meatus is the continuation of the cartilage. Lobule portion of the pinna does not have cartilage.

#### *External auditory meatus*

This canal is a 25mm long, tortuous, cartilaginous bony canal located between tympanic membrane and pinna. Outer cartilaginous part of the canal is directed inwards, upwards and backwards while inner bony part of the canal is directed inwards, downwards and forwards. Superiorly, the fibrous cartilaginous part is deficient in the canal.

The auditory canal contains hair follicles and many sebaceous and ceruminous glands. The Fibro cartilaginous portion of the canal secretes cerumen (Wax) and it prevents the foreign body entry and debris[17].

***Tympanic membrane***

The tympanic membrane is an elliptical shaped semitransparent membranous partition which is gray in color. It separates the tympanic cavity from the external auditory meatus. It measures 8-9 mm horizontally and 9 – 10mm vertically with concavity in the external aspect[18]. In adults, it forms 40 - 50° angle with floor and in infants it is horizontal in position. It is convex inward and the central portion is a broad cone called umbo. Umbo is connected with the manubrium of malleus.

***Nerve and Blood supply***

External ear is innervated by greater auricular nerve, lesser occipital nerve, auricular branch of vagus nerve, auriculo-temporal branch of the trigeminal nerve ($5^{th}$ cranial nerve), facial nerve ($7^{th}$ cranial nerve), tympanic branch of the glossopharyngeal nerve.

External ear gets its blood supply from the superficial temporal artery, the posterior auricular artery and the deep auricular artery and the venous drainage by corresponding veins.

**Middle ear**

Middle ear consists of Tympanic cavity, Ossicular chain, Eustachian tube, Antrum and Mastoid air cells.

***Tympanic cavity*** is a mucous membrane lined space, filled with air and it is irregular in shape about 1 – 2 cm inside the temporal bone[18]. It lies in between the osseous labyrinth and tympanic membrane. This cavity measures about 2 – 6 mm transversely and 15mm vertically and has a biconcave shape. This cavity is a six walled space and has three spaces namely, epitympanum, mesotympanum and hypotympanum.

***Ossicular chain***

Tympanic cavity has three auditory ossicles viz. malleus, incus and stapes. Malleus has a head, small neck, lateral and anterior process and small handle. The head lies in epitympanum and the body of incus articulates with the head of malleus. Manubrium suspends the handle with the tympanic membrane.

Incus has a body, short and long processes. The lower end of the lenticular process connects with the head of the stapes. The smallest bone of the body is the stapes. It has a head, anterior and posterior crus and a footplate. The footplate fits into the oval window.

A large air filled space which lies in the upper part of the mastoid is called as Mastoid antrum. It communicates with aditus, which is an opening through which it is connected to the superior part of the middle ear. The mastoid has more number of honey comb type air cells and it helps to decrease the weight of the skull bone.

***Muscles of middle ear***

Two intra tympanic muscles present in the middle ear are Tensor tympani and Stapedius. Tensor tympani attached to neck of malleus and causes stiffness in the tympanic membrane. Tensor tympani is innervated by the mandibular branch of the $5^{th}$ nerve. Stapedius is the smallest muscle in our body and it is connected with the neck of the stapes. Stapedius muscle is innervated by nerve to stapedius which is a branch of seventh cranial nerve (facial nerve).

Stapedius muscle pulls the eardrum anteromedially thereby helps to decrease very loud sounds conducted by the auditory ossicles, so the inner ear escapes from the trauma produced by the noise.

***Nerve and Blood supply***

Middle ear is innervated by Tympanic plexus, which is formed by both the tympanic branch of Glossopharyngeal nerve and sympathetic plexus which is present around the Internal carotid artery.

Middle ear gets its blood supply from the auditory branches of the maxillary artery, and the branches from the Posterior auricular artery. The venous drainage of the inner ear is by the superficial petrosal sinus and pterigoid venous plexus.

**Inner ear**

The inner ear is located inside the petrous part of the temporal bone. The structures inside the inner ear are well protected by the bone, which covers the inner ear and it is the most compact area inside the body. It is the fluid filled compartment and has long cavities and tunnels. Inner ear has a membranous labyrinth and osseous (bony) labyrinth. Membranous labyrinth is present inside the osseous labyrinth.

Some amount of fluid is present in between the bony and membranous labyrinth and this acts like a cushion against the sudden movements of the ear.

***Osseous labyrinth***

The sense of the organ of hearing and balance is present in the osseous labyrinth. It consists of vestibule, semicircular canals and the cochlea. The vestibule is an irregularly arranged ovoid bony cavity which lies in the center of the labyrinth. Oval window lies lateral to the vestibule. Medially two recesses are present. The spherical recess contains the saccule and the elliptical recess containing the utricle.

Endolymphatic duct passes through the opening of the aqueduct of vestibule which is present below the elliptical recess. Semicircular canal openings are present in the postero-superior part.

***Semicircular canals***

There are three semicircular canals, superior, posterior and lateral. All 3 canals are arranged in such a manner that they lie at right angles (90°) to each other.

There is a dilatation in one side of the canal called the ampulla which open into the vestibule independently as five openings. The non dilated ends of superior and posterior canals form crus commune, which is a common canal.

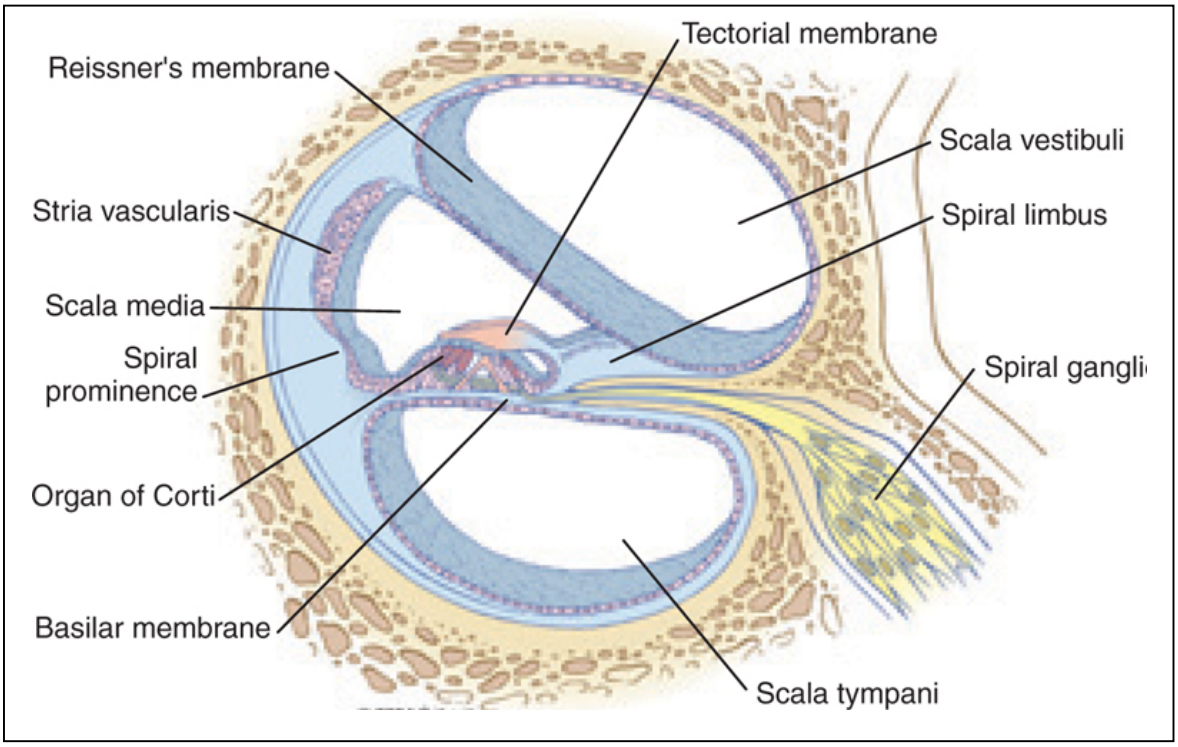
***Cochlea***

The bony cochlea is a coiled shaped tube. It makes 2 ¾ turns (2.5 to 2.75) round the modiolus which are a pyramid like bone[19]. The base of the modiolus is directed internally towards the internal acoustic meatus. The blood vessels and nerve fibers pass through the modiolus to the cochlea.

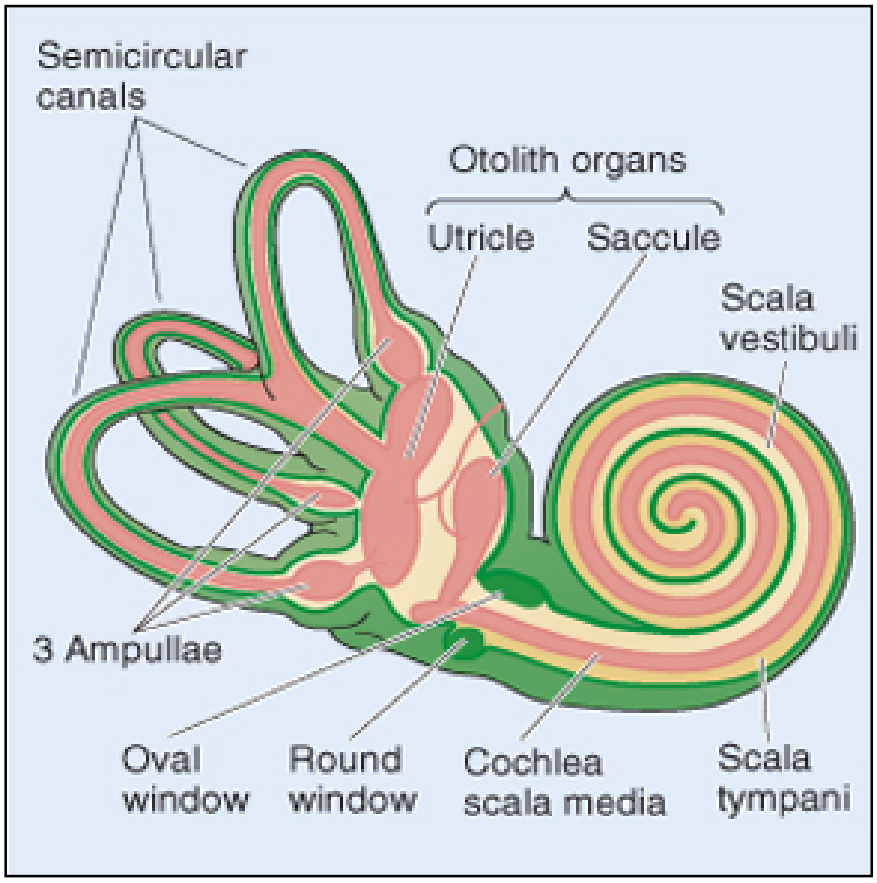
The osseous spiral lamina is a thin plate of bone, which winds round the modiolus like a screw thread. It connects with the basilar membrane and incompletely divides the bony cochlea.

Promontory is a bony bulge present in the medial wall of the middle ear. The bony cochlea consists of three parts, (a) Scala vestibuli, (b) Scala tympani and (c) Scala media (or) membranous cochlea. At the apex of cochlea there is an opening called Helicotrema. Through which scala tympani and scala vestibuli communicate with each other and both are filled with the fluid called perilymph.

## SECTION THROUGH A TURN OF THE COCHLEA




## MEMBRANOUS LABYRINTH



Footplate of stapes closes the scala vestibuli which divides it from the middle ear. Secondary tympanic membrane closes the scala tympani. Aqueduct of cochlea connects the scala tympani with the sub arachnoid space.

***Membranous labyrinth***

Membranous labyrinth consists of cochlear ducts, three semicircular ducts, the saccule and utricle and the endolymphatic sac and ducts. Cochlear duct is a coiled tube with one end closed. In cross section it appears like a triangle. It has three walls, which are formed by Reissner's membrane, Basilar membrane and the stria vascularis. Ductus reuniens connect the saccule and cochlear ducts. The basilar membrane length increases towards the apical coil from the basal coil.

Because of this reason, lower frequency sounds are heard at the apical coil and the higher frequencies are heard at the basal coil. Saccule communicates with the utricle through the saccular duct. The macula is the sensory epithelium present in the labyrinth. Two ducts from the saccule and utricle unite to form the endolymphatic duct and sac.

***Organ of Corti***

The basilar membrane measures 32mm long[20] and 40-80µm width at the base and 500 µm at the apex. It is a fibrous plate contains radially arranged different lengths of collagen fibers. In the under surface, it is covered with mesoepithelial cells.

Organ of Corti is located on the basilar membrane and it is the important sense organ of hearing. It consists of hair cell (sensory receptors) and the supporting structures like supporting cells, tunnel of corti, basilar membrane and tectorial membrane which response to acoustic energy. The receptor cells important for hearing are the hair cells[20]. There are two types of cells, outer hair cells and inner hair cells. These receptor cells convert the sound energy into electrical energy.

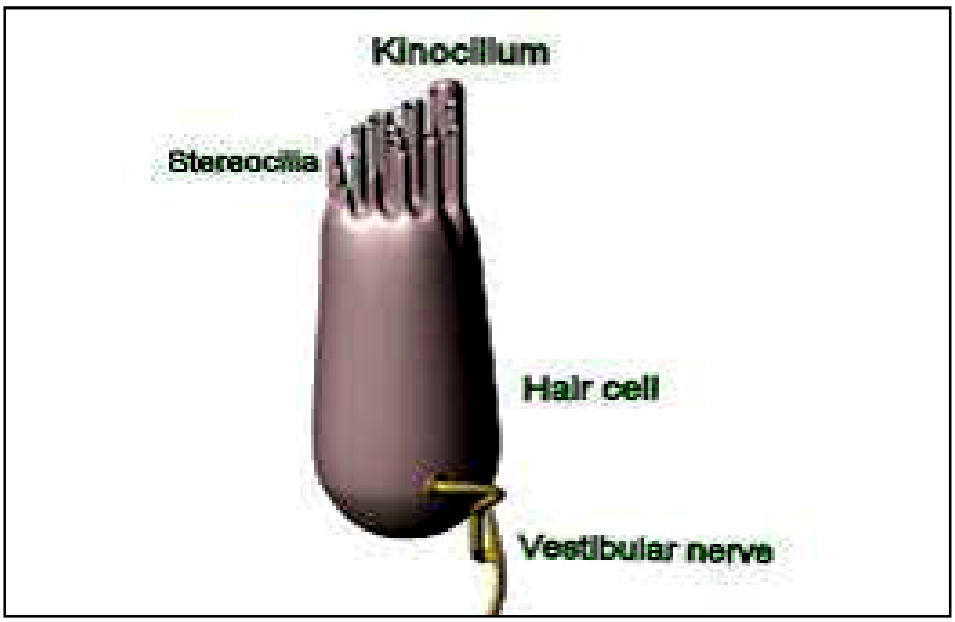
Rods of corti or pillar cells are the rod like epithelial cells arranged in two rows in the organ of corti. Inner and outer rods join at their base to form a tunnel of corti with the basilar membrane. This tunnel is filled with a fluid called cortilymph which is different from perilymph and endolymph. About 4000 rods are present in the corti. The outer pillar rests on basilar membrane and inner pillar rests over the osseous spiral lamina.

There are 3500 inner hair cells present inside the tunnel of corti and arranged in a single row. The main function of these inner hair cells is to transmit the auditory stimuli to the nerve fibers. They are more resistant to damage from the ototoxic drugs and high intensity noise. Afferent cochlear fibers supply the inner hair cells and is important for the auditory impulse transmission. About 30 to 100 rods shaped process or hairs projecting from the apical end of hair cells which contain the protein actin[21]. The afferent neurons contact with the basal end. The inner hair cells have fewer coarser hairs and more rounded in shape.

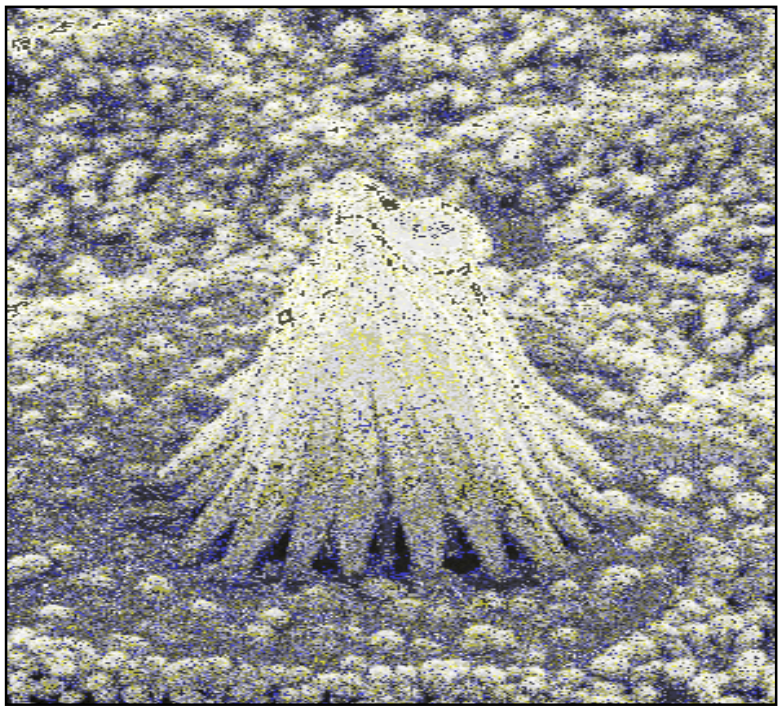
There are 20000 outer hair cells are present in the lateral side of the tunnel of corti and arranged in 3 to 4 rows.

They are cylindrical in shape. They rest on the basilar membrane. There are about 60 to 70 hairs, and each hair is about 4 μm long and are located in the upper end of the outer hair cells. The efferent innervations from the olivary complex are received by the outer hair cells and they modulate the functions of the inner hair cells. They are easily damaged by the high intensity noise and ototoxic drugs. Between the first row and pillar cells, and the last row and the pillar cells, there are

## ANATOMY OF A HAIR CELL




## SCANNING ELECTRON MICROGRAPH OF PROCESSES ON A HAIR CELL



some fluid filled spaces called the spaces of Nuel which surrounds the outer hair cells[19]. Lamina reticularis is a stiff cuticle which covers the hair cells and the supporting cells.

***Structure of hair cells***

The inner and outer hair cells are anatomically similar in their structure except that the inner cell is smaller than the outer hair cell. The hair cell is like a cup shaped structure and its apex has fine hairs (cilia). The cilia are called as Stereocilia. The basal end has synaptic connections with afferent fibers. The stereocilia are about 4 to 10 µm in height and 0.2 to 0.8 µm in diameter and the base of the cilia is narrow[22].

The bases of the stereocilia are attached with the apex of the hair cells. About 50 to 100 stereocilia are present in the apex of the hair cells in a 'W' shaped manner. Towards the stria vascularis the row of the stereocilia is tallest. From the base towards the apex of the cochlea the stereocilia progressively increases in size.

Each stereocilium is closely packed with the cross linked actin filaments with surrounding myosin molecules. The tips of cilia are connected to each other by small strands called tip-links.

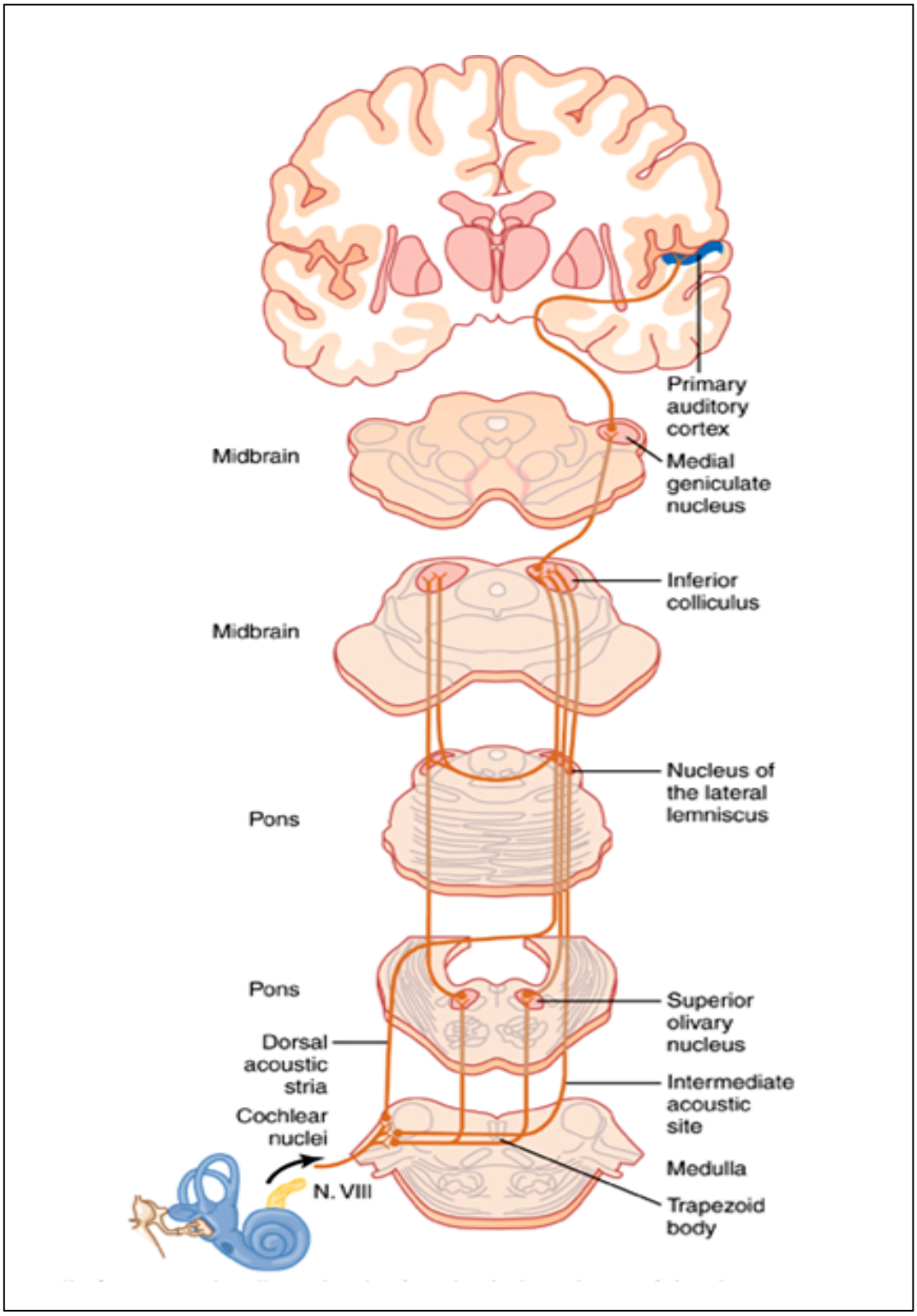
Transduction channels which are mechanically sensitive cation channels, situated at the junction of cilia with the tip-links. From the base towards the apex of the cochlea, the size of the outer hair cells progressively increases.

There are three types of supporting cells present in the organ of corti. They are Deiter's cells, Claudius cells and cells of Hensen. Deiter's cells are located between the hair cells. Cells of hensen and cubical cells of Claudius are situated outside the deiter's cells. Their main function is to provide support to the hair cells. There is an eminence called the Limbus spiralis on the osseous spiral lamina. Its inner margin is attached to the Reissner's membrane. The tectorial membrane contains delicate fibers with gelatinous matrix and it is attached to the outer lip of the limbs spirals. Inner hair cells are in contact with the membrane, whereas outer hair cells are embedded in the tectorial membrane.

***Nerve supply***

The inner ear is innervated by cochlear division of vestibulo- cochlear nerve, which is also called as Auditory nerve. The bipolar cell bodies of the spiral ganglion are located in the

# AUDITORY PATHWAY



canal of modiolus. From the spiral ganglion, the peripheral processes travel through the cochlear canal over the basilar membrane and produce three groups of nerve endings at the hair cell base. They are the external and internal spiral fibers and radial fibers which supply the outer hair cells and inner hair cells respectively.

**Auditory pathway**

Bipolar cells of the spiral ganglion form the first order neurons, which are located on Rosenthal's canal. Dentrites of these cells form the afferent fibers of hair cells. Axons of these cells end in the cochlear nuclei in the medulla.

From the cochlear nuclei, second order neurons pass medially in the dorsal aspect of the pons. The majority of the fibers crosses and go to the opposite side, whereas few fibers remain uncrossed. Most of the crossed fibers of both sides constitute the trapezoid body and some of them do not. All fibers travel to reach the superior olivary nucleus .

From the superior olivary nucleus the fibers ascend as lateral lemniscus and form the nucleus of the lateral lemniscus. The fibers arising from the nucleus terminate in the inferior colliculus.

Fourth order neurons arising from the inferior colliculus reach the anterior brachium of the medial geniculate body. Fibers from the medial geniculate body form the acoustic radiations and reach the auditory cortex. The auditory fibers have multiple decussation points.

**Efferent (descending) fibers**[23]

The afferent fibers descend from the ***auditory cortex*** of the temporal lobe go to (a) Medial geniculate body as cortico-geniculate fibers and also to (b) Inferior colliculus as cortico-collicular fibers.

Fibers project from the ***inferior colliculus*** go to (a) cranial nerve nuclei and to (b) anterior horn cells of spinal cord via spino-tectal tract and also to (c) superior olivary nucleus. Most of the fibers descend in the same side and few fibers cross and go to the opposite side.

From ***olivary nucleus*** the fibers go to (a) bases of outer hair cells of cochlea. Most of the fibers come from the opposite side through the auditory nerve as olivo-cochlear fibers, (b) to vermis of the cerebellum as olivo-cerebellar fibers and to (c)cranial nerve nuclei. The cochlear nucleus is not only a relay station, but also can modify the auditory impulses by converging and mixing the impulses from several regions. But this nucleus only receives the impulses and it indirectly project to the cochlea via olivary nucleus by olivocochlear bundle.

The fibers from temporal lobe also project to the frontal lobe (Frontal eye field, area 8) which is involved in forming the gaze towards the sound originated.

**Inner ear fluids**

Perilymph contains more $Na^+$ ions and resembles the extracellular fluid. It is a blood serum filtrate and formed from the capillaries of spiral ligament and some researchers said that it is the direct continuation of the cerebro-spinal fluid. Endolymph has more $K^+$ ions and resembles the intracellular fluid and fills the membranous labyrinth. Stria vascularis of cochlea secretes the endolymph.

### Development of the ear

Tragus is developed from the first arch tubercle. Rest of the pinna is developed from the second arch tubercle. The external auditory canal is developed from the first bronchial cleft and formed completely at the end of the 28th week of intrauterine life[24]. The tympanic membrane is developed from ectoderm, endoderm and also from the mesoderm. The turbo – tympanic recess of the first and second pharyngeal pouches joined together to form the middle ear cavity. Malleus and incus are derived from the First pharyngeal arch and the stapes is developed from the second pharyngeal arch, but the footplate of stapes is developed from otic capsule.

Inner ear develops from 3rd week of uterine life and completes by 16th week. All inner ear contents are developed from auditory vesicle.

## Physiology of hearing

### External ear

In humans, pinna is a flat cartilaginous flange with a dip in the centre and a raised rim in the periphery called concha. From the external environment, the pinna funnels the sound and sent into the ear due to its peculiar shape.

The pinna also has an important role in the localization of sound from the surroundings in the vertical and horizontal plane, i.e. to know the direction of the sound either from the front or back and from above or below.

External auditory meatus helps the sound waves to reach the tympanic membrane and produces specific resonance frequencies for different sounds. The resonant frequency of External auditory meatus is about 3000 Hz and the concha is around 5300 Hz[25]. The main function is to conduct the sound to the middle ear.

There is 15dB increase in the sound over the frequency range of about 1.5 to 7 kHz while travelling through the pinna and the External auditory meatus. It is mainly because of a) the resonance of the concha around 5 kHz and b) the resonance of the external canal around 2.5 kHz. Binaural interactions are the powerful cues for the localization of sound. External ear provides cues for only monaural localization whereas inner ear provides cues for both monaural and binaural localization.

## Middle ear

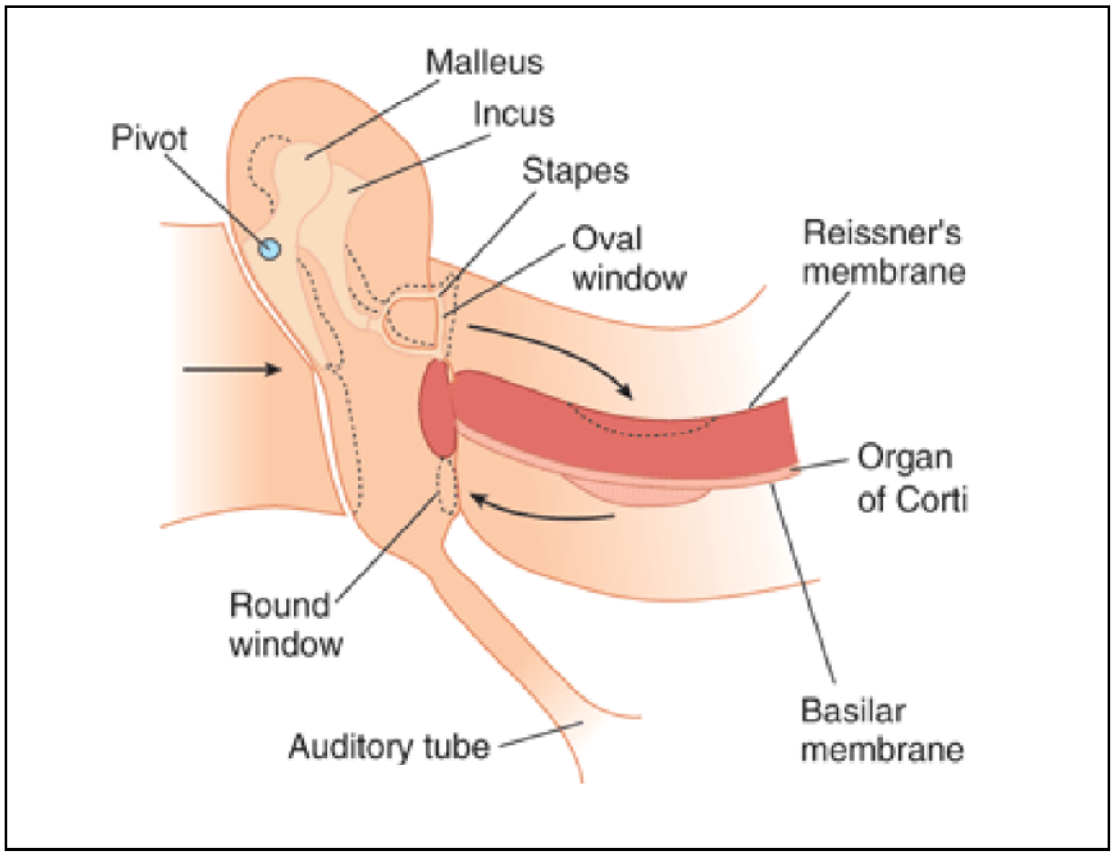
Tympanic membrane acts as a resonator and it is very sensitive to changes in pressure. It stops the vibration whenever the sound wave stops. In the center of the tympanic membrane the handle of malleus is attached. Tensor tympani muscle constantly pulls the attachment and keeps the tympanic membrane in tension and allows the vibrations of sound on any portion of the membrane to be conducted to the ossicles. The other end of malleus is attached with the incus by small ligaments and acts as a single lever. The border of the tympanic membrane acts as a fulcrum for this lever.

The incus is attached to the body of stapes and this causes the footplate of stapes to push forward over the oval window like a door. The combined action of all these ossicles is called as ossicular coupling.

### *Impedance matching of the ossicular chain*

Impedance matching is the important function of the middle ear. The middle ear transfers the comparatively large and low impedance vibration coming from the tympanic membrane to the

# AUDITORY OSSICULAR SYSTEM



much smaller and higher impedance oval window. The middle ear is an excellent impedance transformer. This will change low pressure high vibrations into high pressure low displacement vibrations suitable for cochlear fluids.

The impedance of the cochlear fluids and the sea water is nearly equal (i.e. 1.5 x $10^6$ N.sec/$m^3$). The ossicular chain system reduces the distance and at the same time increases the force of the movement. The impedance matching mechanism involves two processes of the middle ear.

1) The area of the tympanic membrane is 55mm² and is larger than the footplate of stapes which is average of 3.2 square millimetre[26]. The forces coming from the ear drum are exerted over a smaller area, and thus increases the pressure over the oval window of about nearly 17 times (55/3.2 =17.19).
2) The second mechanism is the lever action by the ossicles. The malleus is longer than the incus and this causes a lever action and increases the force by 2.1 times. The velocity at the stapes decreases by 2.1 times.

The total force acting on the cochlea is determined by multiplying these two factors and this is around 22 times (17.19 * 2.1 = 22.34). Thus the secular system and tympanic membrane cause the impedance matching between the sound waves in the fluid of the cochlea and the sound waves in the ear. Usually it is about, 50 to 75% of the frequencies of sound between 300 and 3000Hz transmitted into the cochlea. If the tympanic membrane and the ossicular chain are absent, then the sensitivity of hearing will decrease about 10 to 20dB.

***Influence of ear muscles in the attenuation of sound***

When the loud sounds are conducted through the ossicular chain, after a latent period of about 40 to 80 milliseconds, a reflex occurs to cause contraction of the stapedius muscle and the tensor tympani muscle to a lesser extent. This is called as ***attenuation reflex or tympanic reflex***. The tensor tympani muscle causes the inward movement of malleus and the stapedius muscle causes the outward movement of stapes[27]. This opposite force makes the entire ossicular chain to become rigid and move closer. So the vibrations transmitted to the inner ear is greatly reduced, mainly the low frequency sounds below 1000Hz[28, 29].

The attenuation reflex can decrease the intensity of the low frequency sounds of about 30 to 40dB. The importance of this function is,

1. To protect the cochlea and inner ear structures from the damaging vibrations produced by excessive loud sound.
2. In loud environments, the reflex masks the low frequency sounds. This permits the human voices to be audible even in noisy environment.
3. To minimize the hearing of one's own voice. Because the muscles are active during and before the vocalization.
4. It also suppresses the movements produced by swallowing, chewing and walking. This effect is mainly due to the activation of collateral nerve signal transmission to these muscles and the brain controls the voice mechanism.

***Air conduction and bone conduction***

Conduction of sound waves via the tympanic membrane and the auditory ossicles to the fluid of the inner ear is called *ossicular conduction or air conduction***.** Another type of conduction, *bone conduction***,** is also present and it is the

transmission of sound vibrations to the fluid of the inner ear through the bones of the skull. Bone conduction occurs, when any vibrating bodies like tuning forks are placed directly over the skull. This route is used in the transmission of extremely loud sounds.

***The cochlea***

This is a snail shaped fluid filled part of the inner ear. It has the bony cochlea and membranous cochlea (cochlear duct). The cochlea has three fluid filled compartments viz., scala vestibuli and scala tympani which are filled with perilymph, and scala media which are filled with endolymph.

Stria vascularis secretes the endolymph which is present along the wall of the scala media. The secreting cells of stria vascularis are classified into marginal and basal cells. The stria vascularis pumps out the $Na^+$ ions and pumps $K^+$ ions inside the scala media. The cells in the endolymph have a high concentration of $Na^+K^+$ATPase, carbonic anhydrase and adenylyl cyclase. So the potassium level is high and the sodium level is low in endolymph.

But the electrical potential inside the cells range from +50 to +120mV with respect to plasma. Johnstone and Sellick reported that, the electrical potential of scala tympani (+7mV) is more positive than scala vestibule (+5mV)[30]. The thin Reissner's membrane moves easily and it never obstructs the passage of sound vibration from scala vestibule to scala media.

***Sound transmission in cochlea***

The sound waves from the external environment is transmitted by the eardrum and ossicular chain and cause the movement of the footplate of stapes. The movement of the foot plate of the stapes produces the travelling waves in the perilymph of the scala vestibuli. The height of the wave increases to a maximum, when it moves towards the cochlea, then drops off rapidly. The distance from the stapes to the point of maximum height varies with the various frequency vibrations of the initiating wave. High-pitched sounds produce waves, which reach the maximum height near the base of the cochlea and the low-pitched sounds produce waves, which peak near the apex.

The basilar membrane is depressed into the scala tympani by the waves in the scala vestibuli, because the membrane is not under tension. The fluid displacements in the Scala tympani are dissipated into the air at the round window. So the sound causes distortion of the basilar membrane. The frequency of the sound wave determines the site of maximal distortion.

In the organ of Corti, the reticular lamina holds the top of the hair cells, as rigid with cross links. So that the hair cells move as a stiff bundle.

When the stapes moves, both the tectorial membrane and basilar membrane move in the same direction, but they are attached in different axis, so the outer hair cells bend when a shearing motion hit the cochlea. The inner hair cells are bent by the fluid moving between the inner hair cells and the tectorial membrane. Because the top of the inner hair cells is not attached to the tectorial membrane. The elastic tension produced by the bending of basilar fibers causes the fluid wave in the helicotrema.

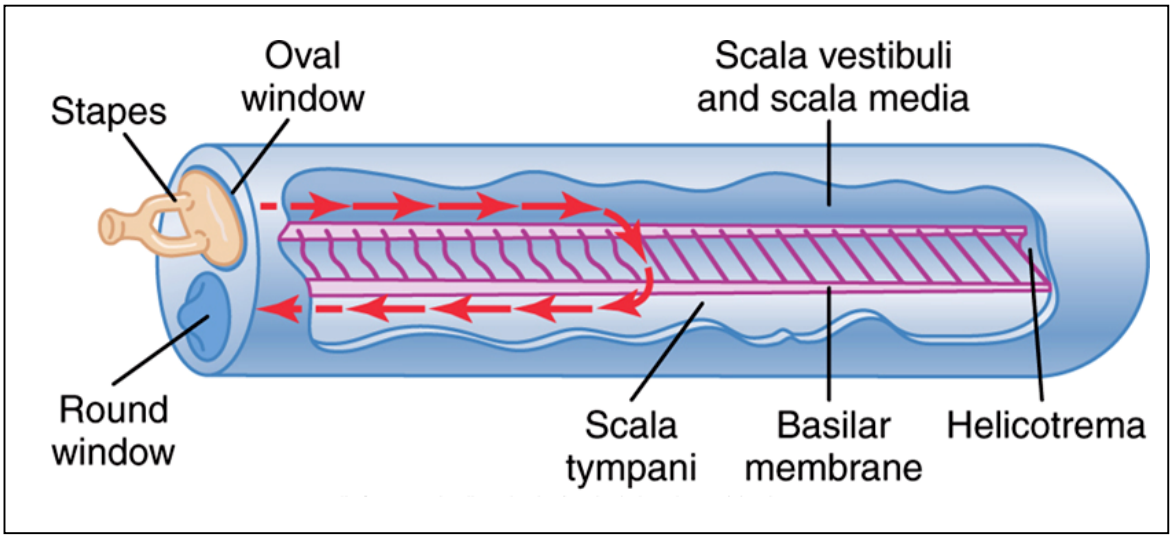
**Transduction by hair cells**

The stimulus is coupled directly to the mechano electrical transduction channels of hair cells[31]. The action potential frequency of the single auditory nerve fiber is directly proportional to the strength (loudness) of the sound stimuli. The individual axon discharges only one frequency sound at low sound intensities, and this frequency different from axon to axon which depends on the origination of the fiber in the cochlea.

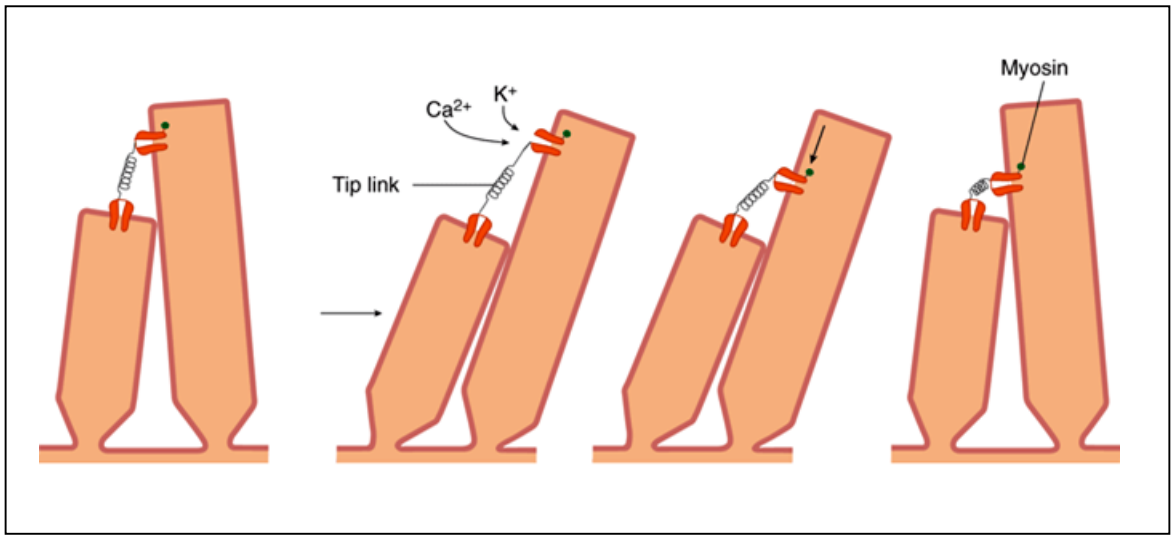
Each axon discharges wider spectrum of sound frequencies at higher sound intensities, especially for the frequencies which are lower than that, at which threshold stimulation occurs.

The hair cells are mechano receptors and they are sensitive to the degree and direction of movement of cilia. Lateral bending of the cilia is the stimulus for these receptors. Even a deflection of 0.5mm, produces a response in the hair cells. When the bundle of stereocilia is stimulated, different rows of stereocilia slide in relation with one another.

## MOVEMENT OF FLUID IN COCHLEA AFTER FORWARD THRUST OF THE STAPES




## ROLE OF TIP LINKS IN THE RESPONSES OF HAIR CELL



The shorter stereocilia bend towards the taller stereocilia i.e. towards stria vascularis and they connect at their tops by the fine links which are directed upwards[32]. One of the cause for *hearing loss* is the mutation of gene called Cadherin-23 which is a component of the tip-link[33].

Whenever the cilia are bent by a stimulus, about 200 to 300 cation channels open and allow the entry of most of the potassium ions and calcium ions, into the cells. The increased level of cytoplasmic calcium concentrations leads to the movement and fusion of the vesicles along with synaptic specialization at the base of the hair cells.

This causes the release of glutamate into the synaptic cleft and produce action potentials in the afferent nerve fibers[34]. The increase in the action potentials depends on the release of the neurotransmitter.

A molecular motor made up of myosin pulls the cation channels to the resting state after depolarization and the stretched tip-links will relax[35]. Through the leaky channels, the potassium diffuse along the electrochemical gradient at the basolateral membrane. If the shorter stereocilia bend away from the taller stereocilia i.e. towards the modiolus, then the apical transduction channels will close and cause hyper polarization.

The resting membrane potential (RMP) of the hair cells is -60mV. The RMP is due to the continuous efflux of potassium ions across its basolateral membrane. Perilymph has zero potential and low potassium concentration in the basolateral surface of hair cells, whereas endolymph has +80 mV potential which is called as endolymphatic or endocochlear potential and high potassium concentration in the apex of hair cells. So the difference in potential across the apical membrane is 140mV [+80 - (-60)]. This large electrical potential gradient causes the cation ($K^{+}$) influx through the transduction channels.

When a sound wave strikes the ear, the organ of Corti is maximally stimulated and it determines the pitch perceived. The travelling wave produces a depression in the basilar membrane, and causes maximal receptor stimulation at one point. The pitch of the sound is inversely related to the distance between the stapes and this point. The nerve fibers respond with an impulse to each sound wave, when the frequency is low. This is called volley effect and it has only limited value[36]. The frequency of the action potentials of the auditory nerve fiber determines mainly the loudness, rather than the pitch.

The pitch of a sound primarily depends on the frequency of the sound and to some extent loudness and duration of the sound. High tones (above 4000 Hz) seem higher and lower tones (below 500 Hz) seem lower, as their loudness increases. To perceive the pitch of a tone, it should last for more than 0.01 s, and the duration should be between 0.01 and 0.1s[36].

**Auditory responses of inner hair cells**

Cochlear nerve endings are emerging from the bases and sides of the hair cells. About 90 to 95% of nerve fibers end in the inner hair cells. But inner hair cells stimulate only 90% of auditory nerve fibers. About 20 afferent fibers terminate in a single inner hair cell. So the inner hair cells are more important in the detection of sounds.

/ The outer hair cells have fewer numbers of synaptic connections with afferent fibers. If these cells are damaged it will cause more hearing loss, even in the presence of normal inner hair cells. From the brainstem more retrograde fibers are emerging and terminate in the outer hair cells. At different pitches, the outer hair cells mainly control the sensitivity of the inner hair cells (tuning of receptor).

The outer hair cells also responsible for the cochlear microphonics. Hyper polarization makes the microphonics lengthier and depolarization makes it shorter. At the same time there is some change in the motor protein called Prestin[36].

Prestin, a transmembrane motor protein, which belongs to the family of SLC26 and is highly expressed in outer hair cells of the mammalian cochlea [37].

**The electrical potentials of Cochlear hair cell**

There are 3 types of electrical potentials. They are,

1. **Endonuclear potentials**: - This is the potential difference between the endolymph and perilymph and it is around 80 to 100mV. It is due to the differences in the composition of the fluids and by the metabolism occurring in the stria vascularis. The difference between the endolymphatic potential and hair cell potential is about 150 to 170mV[38].

2. **Cochlear micro phonic potentials**:- These potentials of cochlea in response to acoustic stimuli is studied by placing electrodes in the scala media and scala tympani. These are AC potentials. There is a DC shift in the micro phonics[38].

Neural potentials are made by a series of deflections at the beginning and at the end of the stimulus in the negative direction. There are 2 phases, longer $N_1$ potential and smaller $N_2$ potential.

Distortion of outer hair cells produces the electrical changes, like the piezo electric potentials produced in the microphone crystal. It is damaged by ototoxic drugs like kanamycin and amikacin and ***also by alcohol***.

3. There are also some **summating potentials** either positive or negative, emerge from the cochlea due to the auditory stimulus. Bending of hair cells produces the positive potentials and the neural fibers cause the negative potentials.

**Theories of hearing**

***Place coding or Place theory of Helmholtz:***

Encoding of specific tones at specific places on the basement membrane is called the Place coding or place theory. Each successive area of the basilar membrane is sensitive to even a slight different frequency. High frequency sounds stimulate smaller fibers at the base of the cochlea and low frequency sounds stimulate larger fibers at the apex of the cochlea.

***Telephonic theory of Rutherfold:***

In this theory, Rutherfold said that basilar membrane is stimulated by every frequency of sound and the cochlea acts as a telephone transmitter.

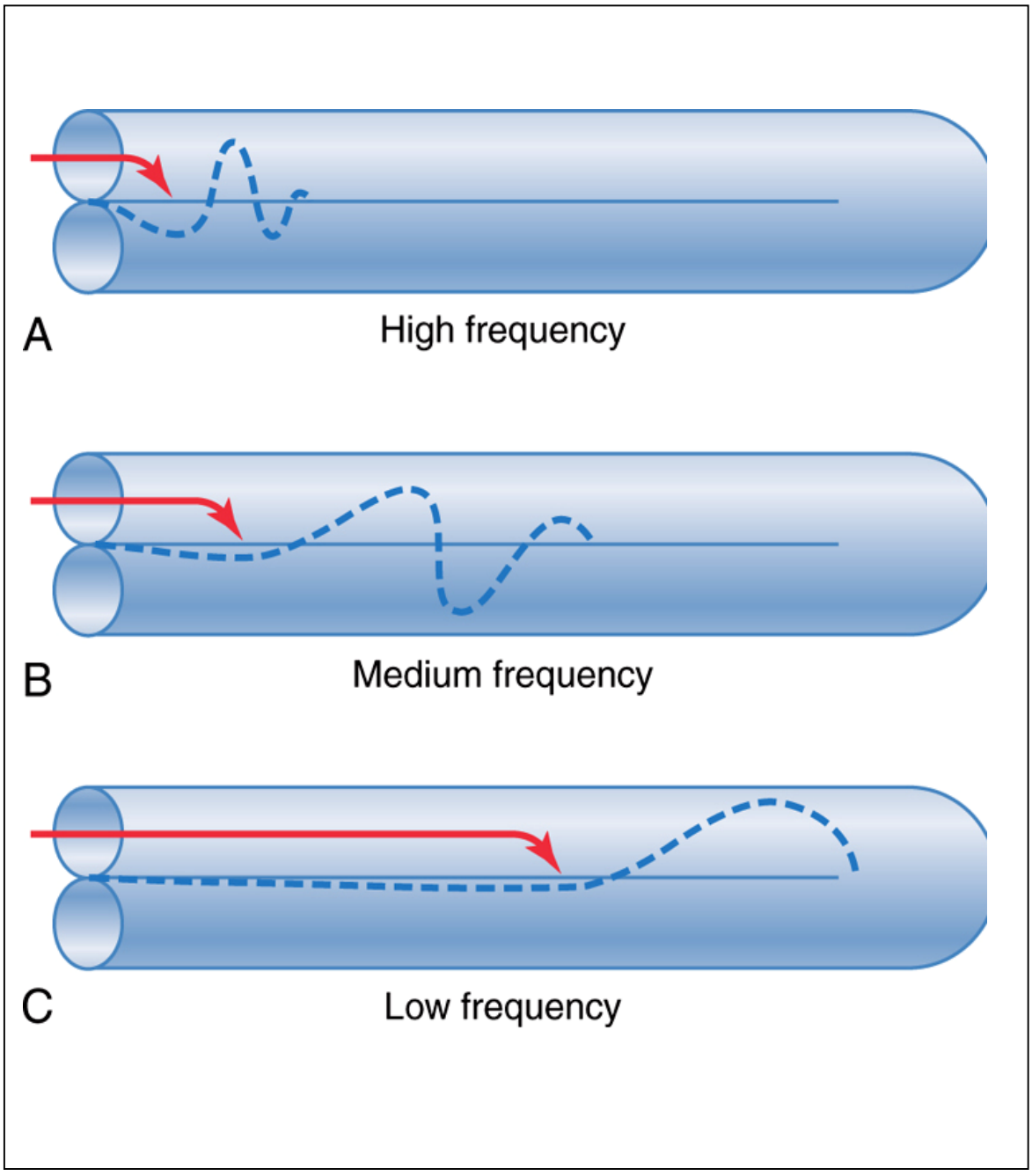
***Wever's volley theory:***

According to this theory, higher frequency sounds (above 5000Hz) stimulate the hair cells present only in the basal cells, but the lower frequency sounds stimulate the entire organ of corti.

***George Von Bekesy's Travelling wave hypothesis:***

In brain, the perception of different frequency tones depends upon the type of wave generated at the basilar membrane and cochlea. According to this hypothesis, the receptors receive high frequency tone at the basal region of the cochlea and low frequency tone at the apex region. This is the most accepted theory.

# Travelling waves along the basilar membrane for high, medium and low frequency sounds



**Characteristic frequencies of cochlear nerve fibers**

When a particular sound frequency stimulate a cochlear afferent fiber and if the fiber discharges maximally, then the frequency is called as the characteristic frequency of that fiber.

***Encoding***

The discharges of cochlear nerve fibers contain the different features of an acoustic stimulus. They are in the form of encoding[34]. Duration is signaled by the duration of neural activity and the intensity is signaled by the amount of activity and also by the number of discharging fibers. The frequency is signalled by the afferent fiber, which discharge with the stimulus for low frequency sounds. This is called ***phase locking***. It also occurs for shorter period sounds than the absolute refractory period of the afferent fiber.

A single fiber cannot discharge if the tone is more than 1kHz, with every cycle. From the activity of an afferent fiber, the Central Nervous System detects higher-frequency information, and every fiber discharges in phase with the stimulus as a group, and signal the frequency of the stimulus. On the basis of this principle the frequency theory of hearing was arrived**.**

The place theory dominates in case of higher frequencies (>5000 Hz), with the CNS interpreting sounds that activate afferent fibers of hair cells at the base of the cochlea, because of being higher frequency.

Thus, while explaining the sound frequency coding (duplex theory) across the range from 20 to 20,000 Hz, we require both the place and the frequency theories.

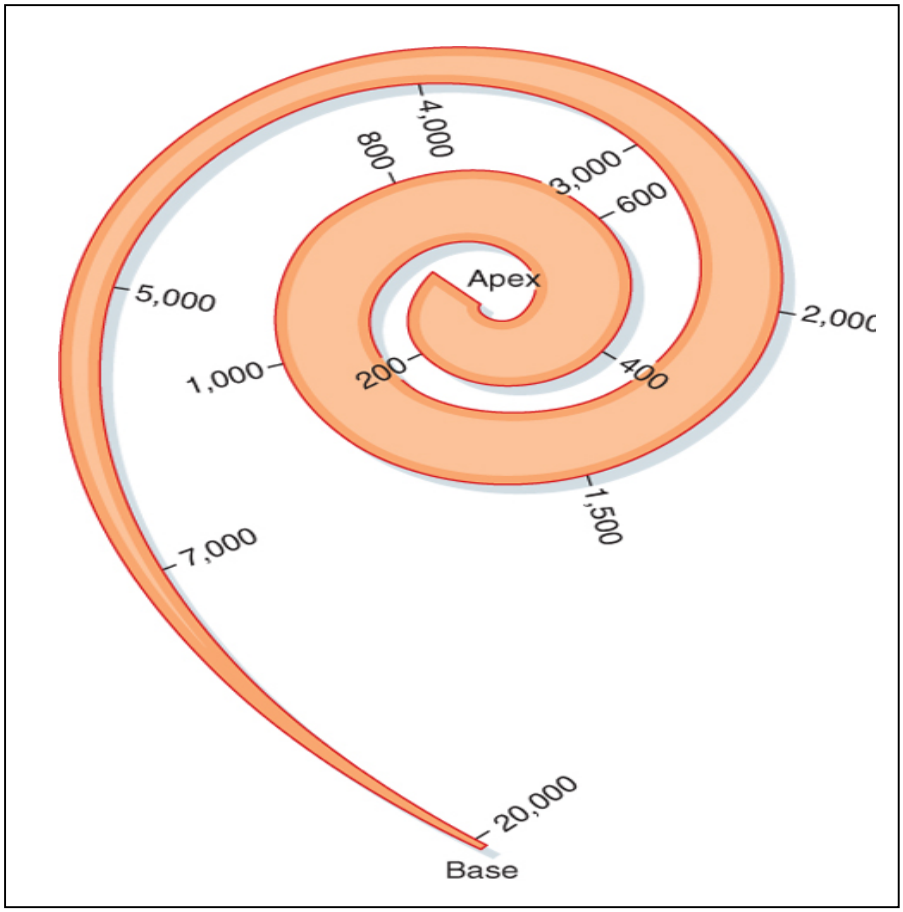
According to Duplex Theory[39], spatial hearing in the horizontal plane transitions between two different mechanisms as the frequency of the sound is increased. Low frequency sounds are localized using inter aural time differences (ITDs), which are created primarily on the basis of differences in path length between each ear and the sound source. High frequency sounds are produced by interaural level differences (ILDs), which are created by a combination of the directional filtering properties of the external ears and the acoustical shadowing effect of the head.

**Organization of the Central auditory system**

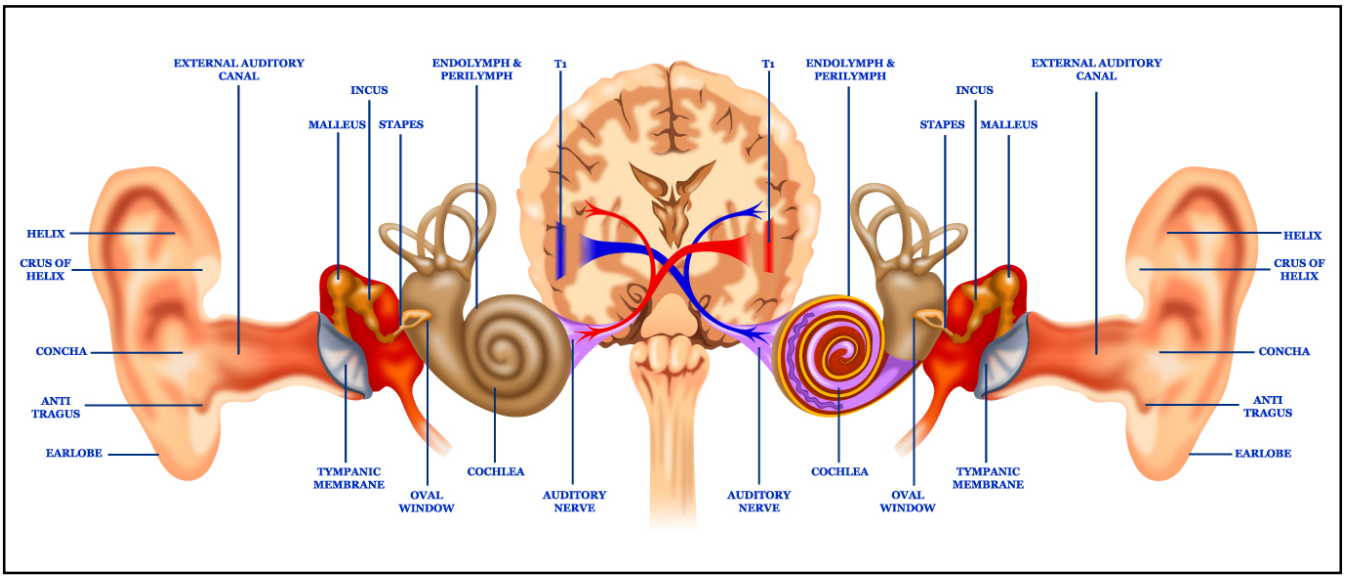
***Receptive fields and tonographic maps***

A tonotopic map is established by plotting the distribution of the characteristic frequencies of neurons in the auditory cortex, and can be found out which neurons are ordered by their best frequencies.

# TONOTOPIC ORGANIZATION




# BINAURAL HEARING



Tonotopic maps have been found in the auditory cortex, medial geniculate body, inferior colliculus, superior olivary complex, and cochlear nuclei. The representation of tone in the cochlear neurons is maintained in the order and also in the neurons of the auditory pathway. This is known as tonotopic organization. Apical region of cochlea respond more to low frequency sound and base responds to high frequency sounds.

***Binaural interactions***

At the level of cochlear nuclei, most of the auditory neurons respond to stimulation of either ear (i.e. They have binaural receptive fields). Sound localization is contributed by the binaural receptive fields. Even with one degree separation of sound sources, a human ear can easily distinguish the separation. The auditory system judge the origin of sounds using several factors. They are the differences in between the time of arrival of the sounds in the two ears and the loudness difference between the two sides of the head.

The above factors provide information regarding the location of the sound by analyzing the activity of the superior olivary complex neurons.

The synapses of the medial dendrites are mostly excitatory, and they emerge from the contralateral ventral cochlear nucleus. The synapses of the lateral dendrites are largely inhibitory and they originate from the ventral cochlear nucleus of the same side. Differences in the phase of the sound coming to the two ears change the strength and timing of the inhibition and excitation reaching the medial superior olivary neuron and it provides the information regarding the sound localization. The lateral superior olivary neuron provides the information about the source of the sound.

Yong Lu et al suggested that in the binaural interactions there was some difference in ipsilateral and contralateral excitatory Frequency Turning Curves (FTCs), in their animal study using big brown bat[40]. Kyle T. Nakamoto et al demonstrated that, nonfocal deactivation of the auditory cortex modifies the sensitivity of number of neurons in the inferior colliculus (IC). This was one of the major cues for the localization of sound in space, interaural level differences (ILDs), in an anesthetized guinea pig[41].

### *Cortical organization*

Primary auditory cortex contains the sensory map, and also the tonotopic map, but this cortex also performs feature extraction. The primary auditory cortex neurons forms isofrequency columns (neurons have the same characteristic frequency), and alternating columns called as summation and suppression columns. Summation column neurons are more responsive to binaural than to monoaural input. Suppression column neurons are less responsive to binaural than to monoaural stimulation and so the response to one ear is dominant[42]. In the guinea pigs, Donald Robertson and Dexter R.F. Irvine demonstrated that in some disorders like partial deafness the auditory cortical frequency map may undergo reorganization[43].

## Hearing loss

Hearing is the most important sensory modality which is necessary for the mental development of a child. For normal speech and language development, verbal speech is necessary. In humans, hearing loss is one of the most common sensory defect. The gradual loss of hearing is associated with aging, affecting those over 75 years, is called as Presbycusis.

It is mostly due to the gradual and cumulative loss of neurons and the hair cells. Hearing loss is a multifactorial disorder in most of the persons and also caused by genetic and environmental factors.

Deafness can be classified into two major categories, conductive (or conduction) hearing loss and sensorineural hearing loss. Conductive hearing loss means the decreased or impaired transmission of sound in the external or middle ear or both and it has an impact on all frequencies of sound. The causes of ***conduction hearing loss*** are, obstruction of the external auditory meatus with wax (cerumen) / any foreign bodies, any inflammation of the outer ear (otitis externa) / inflammation of the middle ear (otitis media) which leads to fluid accumulation, perforation of the tympanic membrane, and otosclerosis in which sclerotic bone grows over the oval window by replacing the resorbed bone.

**Sensorineural hearing loss (SNHL)**

Sensorineural deafness are mostly caused by the loss of cochlear hair cells. It can also be caused by impairment of eighth cranial nerve or central auditory pathway.

It may be congenital or acquired. It usually affects the hearing ability of certain pitches, while others pitches remain unaffected. It can be either total deafness or mild or moderate degree of deafness.

***Causes***

*Congenital causes* include maldevelopment of the inner ear, injury to the hearing apparatus before or at the time of delivery. The acquired causes begin to appear later after delivery. It may be genetic (delayed onset) and cause only hearing loss or may associate with defects in other systems in cases of syndromes like Pendred syndrome. The inner ear ionic homeostasis were affected by several genes[44].

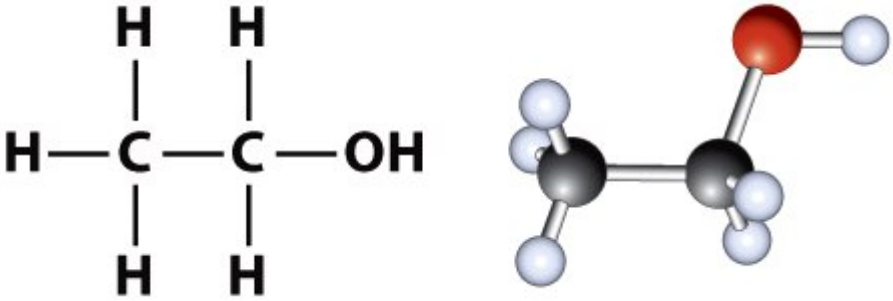
*Acquired causes* of SNHL:

1. Labyrinthial infections due to microorganisms.
2. Injury to the labyrinth or vestibule-cochlear nerve by fractures of mastoid bone or by surgery.
3. Noise induced hearing loss.

4. Ototoxic drugs: - Aminoglycoside antibiotics such as streptomycin and gentamicin, diuretics, cytotoxic drugs and also due to ***drug overdose of alcohol*** and tobacco[24]**.**
5. Presbycusis.
6. Acoustic neuroma.
7. Meniere's disease.
8. Sudden hearing loss.
9. Systemic disorders like diabetes mellitus, hypothyroidism, renal diseases and autoimmune disorders.

**Alcohol an introduction**

Alcohol is one of the organic compound in which the functional hydroxyl group (-OH) is attached to a carbon atom. The term alcohol refers to the ethyl alcohol (ethanol) in alcohol cultures[42]. In alcoholic beverages, it is the dominant alcohol.



Alcohol may be classified as a drug, tranquilizer, sedative, anesthetic or hypnotic, depending upon the quantity used[1]. Self induced intoxication by alcohol is the only socially acceptable drug in western countries. It is rapidly absorbed from the gastrointestinal tract and is distributed throughout the body.

It affects almost all organs and alters the neurochemical processes of the central nervous system.

In western countries, 80% of people consume alcohol, in which 20% of men and 10% of women are having the lifetime risk for serious alcohol problems, regardless of a person's income and education[45].

Alcohol in low doses has few benefits in health systems. Consumption of more than three standard drinks per day increases the risk for liver cancer and vascular diseases. Alcoholic disorders reduce the life span of an individual to approximately about 10 years[45].

**Classification of alcoholics**

Royal college of Physicians, Psychiatrists and General practitioners defined this classification in 1995.

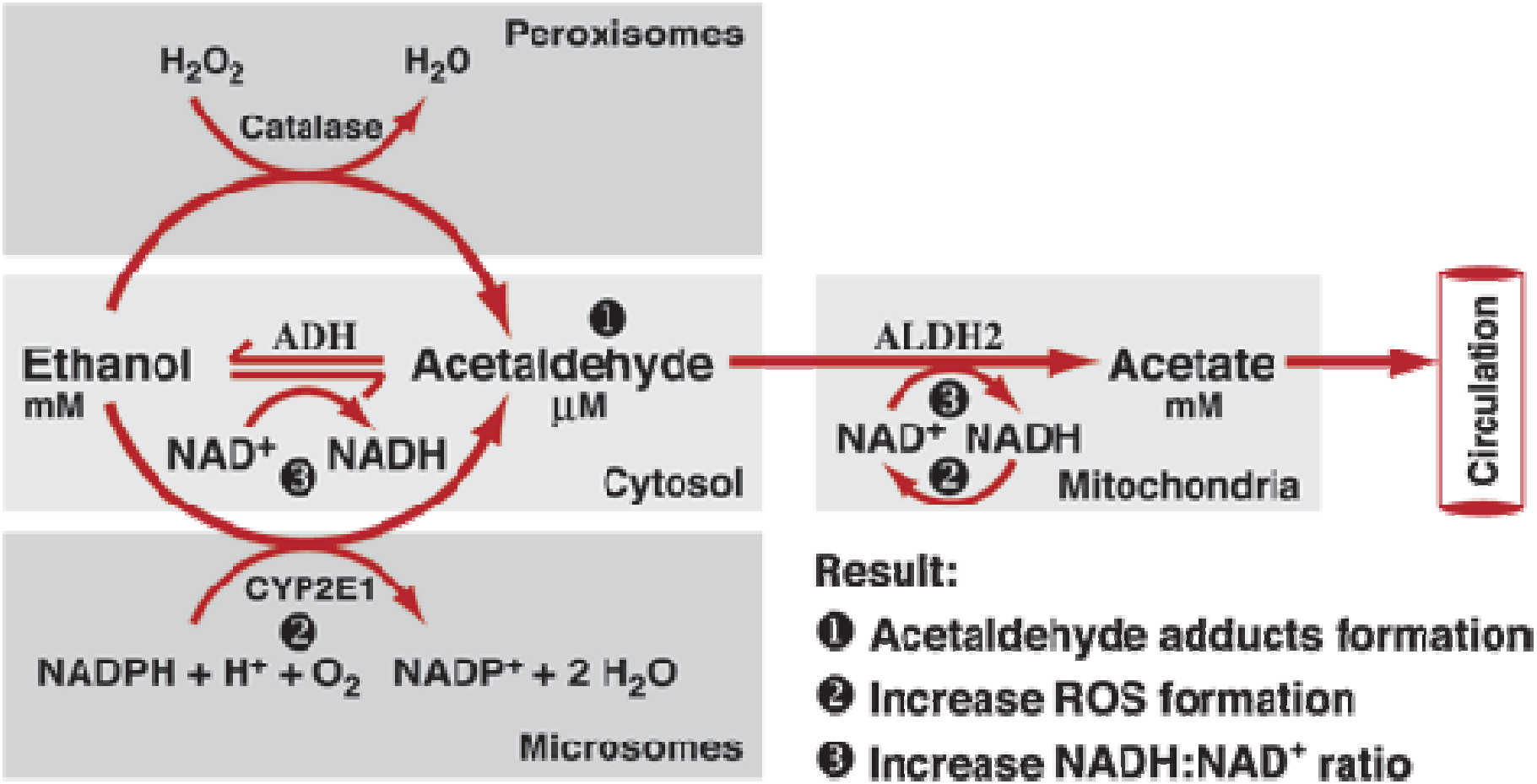
According to this, *Low risk (Insensible)* for men is the consumption of alcohol up to 21 units per week and for women it is 14 units per week (1unit = 8gm of alcohol). *Increased risk (Hazardous)* for men starts from 22 units to 50 units per week and for women it is 15 units to 35 units per week. *High risk (Harmful)* for men is above 50 units per week and for women it is above 35 units per week[3].

According to U.K. standards, the risk of life increases of consuming alcohol $\geq$ 4 units/day for men and $\geq$3 units/day for women. Drinking 8 units of alcohol for men and 6 units for women (Twice the daily limit) is called as Binge drinking[3].

**Identification of alcoholics**[45]

For identification of alcoholics, any one of the following is needed.

1. Six (women) / Eight (men) or more drinks/day consumption of alcohol on a regular basis.
2. Blood tests – Increased Glutamyl Transferase (GGT) of more than 35 units and more than 20 units/L of Carbohydrate Deficient Transformin (CDT).
3. By standard questionnaire (AUDIT – 10 item Alcohol Use Disorder Identification Test) as a screening tool.


Peroxisomes
$H_2O_2$
$H_2O$
Catalase
Ethanol
mM
ADH
Acetaldehyde
µM
❶
$NAD^+$
❸
NADH
Cytosol
ALDH2
Acetate
mM
❸
$NAD^+$ NADH
❷
Mitochondria
Circulation
CYP2E1
❷
$NADPH + H^+ + O_2$ $NADP^+ + 2\ H_2O$
Microsomes
Result:
❶ Acetaldehyde adducts formation
❷ Increase ROS formation
❸ Increase NADH:$NAD^+$ ratio

High normal MCV (≥91μm³) and Serum Uric acid (>7mg/L) are used occasionally as a minor criteria.

**Diagnosis of alcoholism**[45]

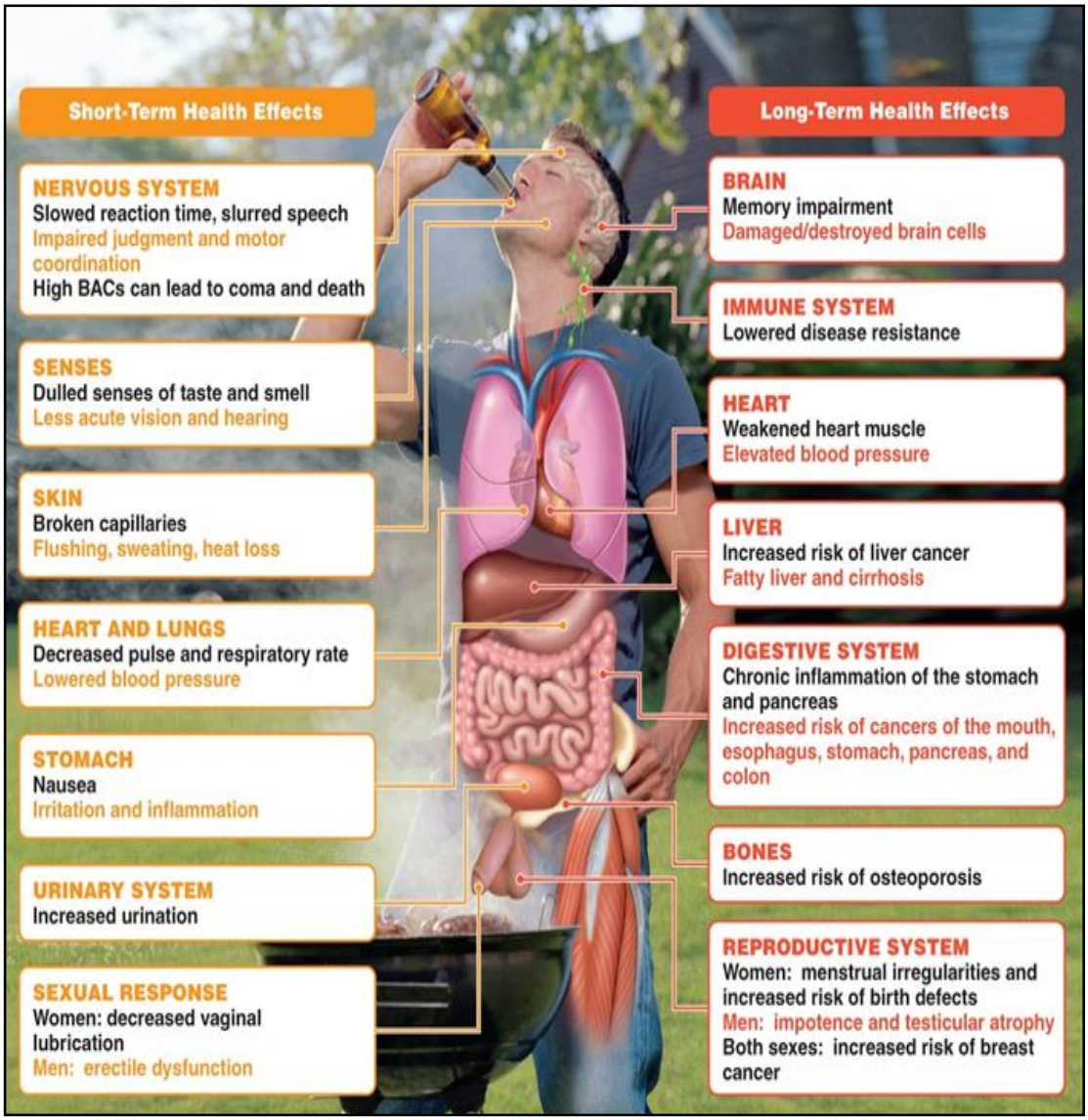
*Alcohol dependence* is defined as the occurrence of repeated alcoholic problems in at least 3 of seven life risk areas that cluster together at about the same time (Over the same 12 month period) according to DSM – IV[46]. Clustering of at least 3 symptoms during the 12 month period is necessary for the diagnosis of alcoholic dependence.

*Alcohol abuse* is defined as having repetitive problems with alcohol in any one of the 4 life areas – Social, Interpersonal, Legal and Occupational or repeated use in hazardous situations which intoxicates the individual who is not an alcohol dependent.

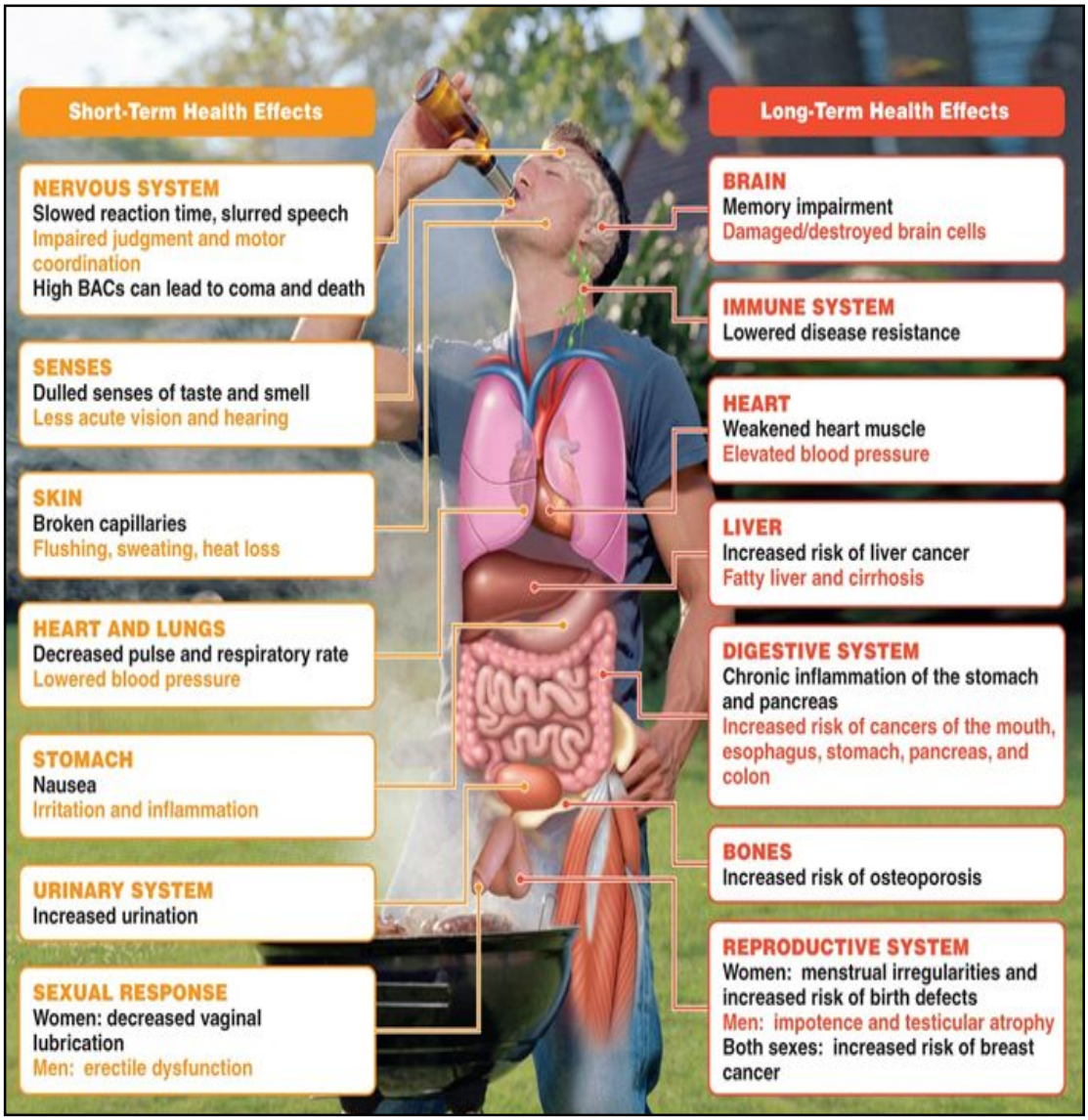
**Mechanism of action of alcohol**

In brain, especially in $GABA_A$ (Gamma amino butric acid type A) receptors, alcohol boost the GABA activity. Inhibition of post synaptic N-Methyl D-Aspartate (NMDA) excitatory glutamate receptors is caused by acute alcoholism while up regulation of these excitatory receptors is caused by chronic alcoholism.

# EFFECTS OF ALCOHOL ON THE BODY AND HEALTH



# EFFECTS OF ALCOHOL ON THE BODY AND HEALTH



**Effects of alcohol on different systems**

Alcohol causes some *beneficial effects* in low doses by increasing High density lipoprotein and decreasing the aggregation of platelets and thereby decreases the risk for embolic stroke and coronary artery diseases[45, 47]. But in high doses, alcohol causes only deleterious effects on all systems.

*Nervous system*

Alcohol causes episodes of temporary anterograde amnesia (Blackout). It reduces the time spent in REM sleep (Rapid Eye Movement) and deep sleep stages. It also causes impaired judgment and coordination. 10 % of Chronic alcoholics who consume high doses regularly will develop peripheral neuropathy.

Cerebellar dysfunction or atrophy develops in 1 % of alcoholics. Korsakoff's syndrome (Retrograde and Anterograde amnesia) and Wernicke's syndrome are also caused by alcohol in very few individuals. Alcohol causes sadness (40%), Temporary severe anxiety (10 – 30%) and auditory hallucinations (3 – 5%).

*Gastrointestinal system*

Alcohol causes Hemorrhagic gastritis, and Mallory Weiss syndrome (longitudinal tear in the mucosa at gastro esophageal junction). Next to the nervous system, it severely affects the hepato biliary system. Alcohol hepatitis and Cirrhosis of the liver are caused by chronic alcohol ingestion. It also causes Alcoholic pancreatitis[48] which results in damages to the pancreas.

*Cancer*

There is a strong pathogenic link between cancer and alcohol[49]. There is 4 fold increased risk of Breast cancer and 1.5 fold increased risk of Esophageal cancer in alcohol consumers. All these effects are due to the cancer promoting effect and indirect interference on immune homeostasis by acetaldehyde and alcohol.

*Cardiovascular system*

Alcohol decreases the myocardial contractility and thereby causes peripheral vasodilatation and decreases in blood pressure and increase in cardiac output by compensatory mechanism. Mild to moderate hypertension is caused by heavy drinking. There is 6 fold increased risk for myocardial infarction and cardiomyopathy. It also causes left ventricular failure and arrhythmias.

*Reproductive system*

In males, alcohol causes decrease in erectile capacity, but increase in the sexual drive. Alcohol produces shrinkage of seminiferous tubules and irreversible testicular atrophy and also decrease in sperm count.

In female, heavy drinking of alcohol causes amenorrhea, decrease in ovary size and infertility. In fetus, it produces fetal alcohol syndrome and decrease in I.Q. (Intelligent Quotient).

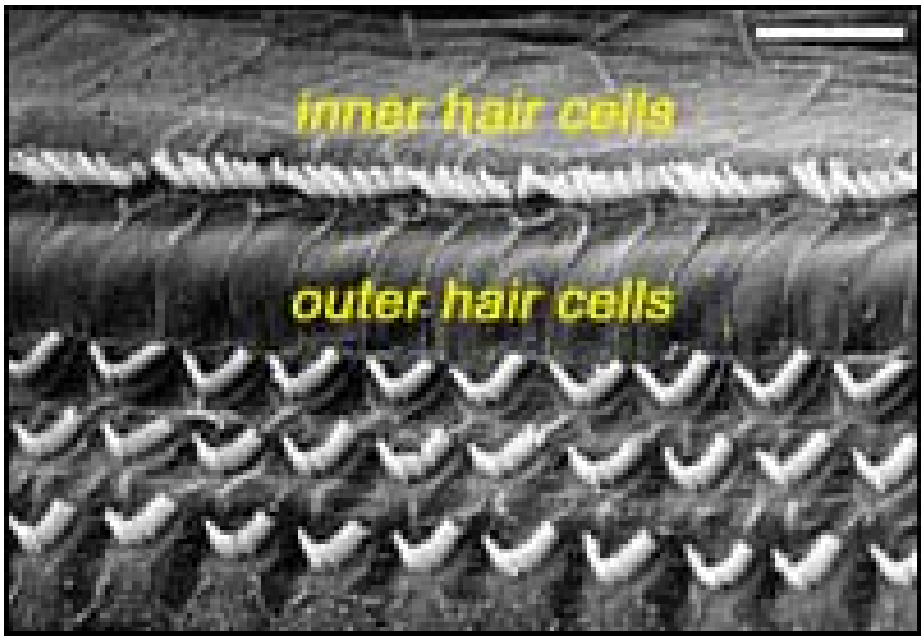
Alcohol also causes myopathy, decrease in the bone density, increase in the cortisol level and also decrease in the level of vasopressin, T3 (triiodothyronine) and T4 (tetraiodothyronine).

*Haemopoietic system*

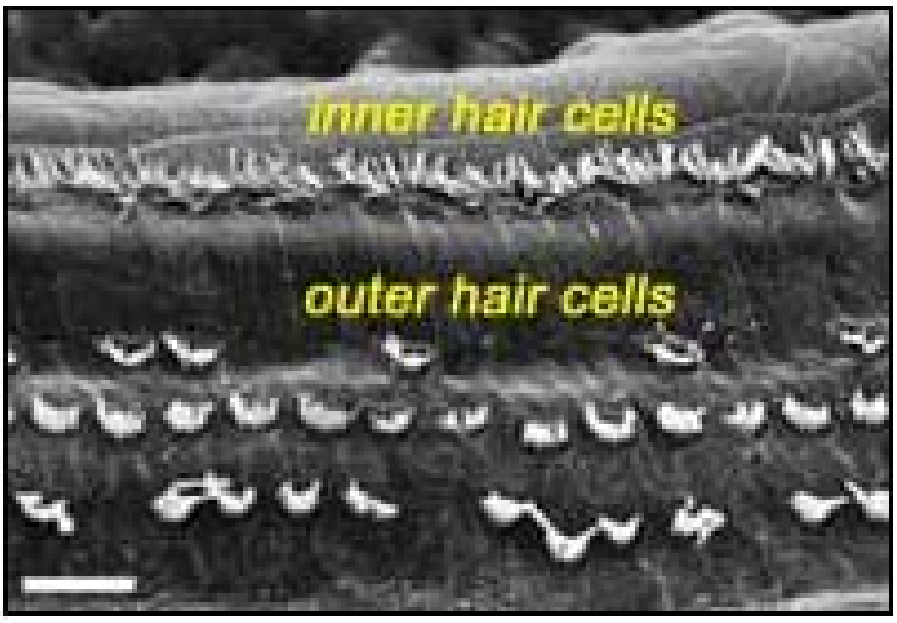
Alcohol causes Reticulocytopenia, Hyperplastic bone marrow and Thrombocytopenia in blood.

**Auditory system**

Alcohol in overdoses affects the auditory system and causes sensorineural hearing loss. Perez R. et al in their animal (sand rat) study demonstrated that alcohol had a toxic effect on the cochlear and vestibular function of the inner ear[50].




Normal




Damaged

***Mechanisms of structural changes***

Alcohol mainly affects the outer hair cells (OHCs) [51,52]. The exact mechanism of alcohol affecting the OHCs remains unknown. There are many possibilities.

Alcohol is one of the ototoxic drug to the outer hair cells. Ototoxic drugs usually affect the OHCs which are present mainly in the basal turns of cochlea. Alcohol causes obstruction in the mechanosensitive channels in the stereocilia of hair cells. It causes the degeneration of cells and produces sensorineural hearing loss.

In this study, Ethanol affects the development of Sensory Hair Cells in Larval Zebrafish (Danio rerio), Phillip M. Uribe showed that, ethanol administration causes increased apoptosis in the CNS of zebrafish[53]. In this study it was concluded that the combination of increased hair cell death and decreased proliferation of supporting cells were the causes for a reduction in the number of mature hair cells in the ethanol treated animals.

Alcohol and its metabolites disturb the endocochlear environment which results in outer hair cell motility variations[51].

Consumption of alcohol over a long period can cause damage to the central auditory cortex of the brain and lead to brain shrinkage[54]. Elisabeth Stephanie Smith said that, brain shrinkage is due to the loss of white matter, and it may partially reversible. Adding damage to the auditory nerves by alcohol, even moderate drinkers have the risk of developing nerve damage and hearing loss.

Alcohol also affects the middle ear muscles and thereby affects the acoustic reflex thresholds in humans. Because, before being received by a microphone in the auditory canal, the DPOAEs must travel through the middle ear. Any change in middle ear muscle tone activity or pressure in the middle ear causes decrease in DPOAE amplitudes.

***Molecular mechanisms***

In the brain, alcohol stimulates the inhibitory effect of GABA activity in $GABA_A$ receptors. Acute alcoholism causes inhibition of post synaptic N-Methyl D-Aspartate (NMDA) excitatory glutamate receptors[55]. NMDA receptors allow the entry of $Ca^{2+}$ ions which activate the second messenger cascades.

Alcohol also suppresses the OHCs by affecting the efferent pathways of cholinergic and GABAnergic[51]. Upregulation of the excitatory receptors is caused by chronic alcoholism[56].

According to the "lipid theory" of alcohol, ethanol produces its effects by acting on the membrane lipids and thereby producing changes in the membrane protein functions[56]. Brain Stem Auditory Evoked Potential latencies were affected by the cumulative effect of lifelong alcohol consumption, which causes damage to the central auditory pathways, that lead to hearing loss. Alcohol also causes delays in the neurotransmission time. Commonly used hearing performance tests cannot detect these defects.

**Recent advances in hair cell regeneration**

Experimentally, Sensorineural hearing loss was successfully treated with Cochlear gene therapy. By using viral vectors atonal homolog1 gene was delivered and used in inner ear hair cell regeneration[57]. This showed that the possibility of cochlear hair cell regeneration. Hair cell regeneration and repair were best studied in the neonatal chick model.

It was shown that, when the damaging cells are induced to proliferate, the new hair cells arose as progeny from the so called non dividing supporting cells[25]. Notch receptor and the basic helix transcription factors have excellent roles in the development and regeneration of hair cells[58].

The cytoskeletal properties and cell-matrix interactions of supporting cells of mice had different ages which gave the explanation for the age-related differences in between mammals and non-mammals hair cell regeneration.

If the hair cells are damaged, spontaneous regeneration of nerve fibers may be seen after some time[59].

Some neurotrophic factors have been proven to induce re-growth in peripheral nerve fiber of animal models. It is associated with enhanced survival of Spiral Ganglion Cells.

**Clinical tests for assessment of hearing**

1. Finger friction tests
2. Lever pocket watch tests
3. Speech(voice) tests

*4. Tuning fork tests*

1) Rinne's test
2) Weber's test
3) Absolute bone conduction tests
4) Schwabach's test
5) Bing test
6) Gelle's test

**Audiometric test**

1. Pure tone audiometry
2. Speech audiometry
3. Bekesy audiometry
4. Impedance audiometry
    a. Tympanometry
    b. Acoustic reflex measurements

**Special tests for hearing**

1. Recruitment
2. Short increment sensitivity index (SISI test)
3. Threshold tone decay test
4. Evoked response audiometry
    a. Electrocochleography (EcoG)
    b. Auditory brainstem response (ABR)
5. Autocratic emissions (OAEs)

**History of audiogram and audiometer**

Arthur Hatmann created an 'Auditory chart in 1885 which included the tuning fork tests of right and left ear in abscissa of the chart and hearing percentage in the ordinate[44]. After that Max Wein designed a 'sensitivity curve' and presented it in 1903. In 1922, Fletcher, Wegel and Fowler analyzed the hearing loss by plotting the intensity in the ordinate and frequency interval in the abscissa and they coined the term 'audiogram'[44]. From 1920 to 1990, the scientists modified the audiogram.

In 1899, the audiometer designed by Carl Seashore was used to measure the 'keenness of hearing'. First commercially based electronic audiometer was introduced by the western electric company in early 1920s. In 1928, the western electric audiometers improved a lot with further advance in the technology with bone conducting testing capabilities. Based on normative values, American Standards Association (ASA) introduced audiometric zero in 1951 (Berlin 1963).

In the end of 1940s, Von Bekesy introduced the automatic audiometry. During the 1960s and 1970s, audiogram with the newer tests like, Tone decay test, Short increment sensitivity index ( SISI test ) and Alternate Binaural Loudness Balance Test (TdT) were introduced.

Nowadays we are using the audiograms based on the American Speech Language Hearing Association (ASHA) standards which was compiled in 1992 and the standards set by the American National Standards Institute (ANSI) in 1996[60].

**Pure tone audiometry**

Pure tone audiometry is an art of detecting the hearing acuity (threshold level of hearing) of a subject for various frequency pure tone sounds. Sound is a form of energy formed by a vibrating object. Frequency is the number of cycles per second and the unit are Hertz (Hz). Intensity is the amplitude of the sound and its unit is Decibel (dB).

Sound Pressure Level of a sound in decibels is 20 times the logarithm to the base 10 of the pressure of a sound, to the reference pressure (0.00024 dynes/sq.cm) [24]. The minimum sound that a healthy subject may hear for a given tone or frequency is called as Hearing threshold. The normal hearing threshold is 0dB that means the minimum sound perceived by 50% of normal healthy persons.

Pure tone is a single frequency sound (e.g. A sound of 250, 500, 1000Hz etc.). The electronic device used to detect the hearing acuity is called a puretone audiometer.

When the results are plotted graphically then it is called a puretone audiogram. Puretone audiometer delivers pure tone sounds of various frequencies from 125 Hz to 8000 Hz to the ear. The normal range of hearing in humans is 20Hz to 20000Hz.

It consists of an audio - oscillator which produces pure tone sounds, a frequency selector, an amplifier, and noiseless switch interrupter and accessories as headphones and bone conduction vibrator. Headphone is used to conduct the sound into the ear canal and bone conduction vibrator is used to deliver the sound through the bone.

While operating the audiometer, the tester should never give any visual clues to the subjects. He also should confirm that the audiometer got the calibrations regularly checked. The headphones are fixed to the subject's ear so that the diaphragm is placed at the entrance of meatus. Care to be taken to avoid the presence of hair in between the ear and transducer and there should be no occlusion of the ear due to pressure over the tags.

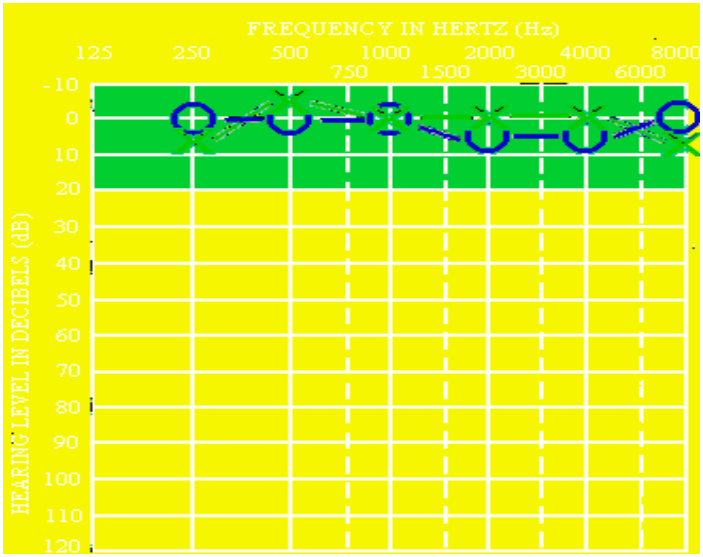
*Air conduction threshold test*

A clinical history and a detailed examination should precede the test. Both ears should be wax free.

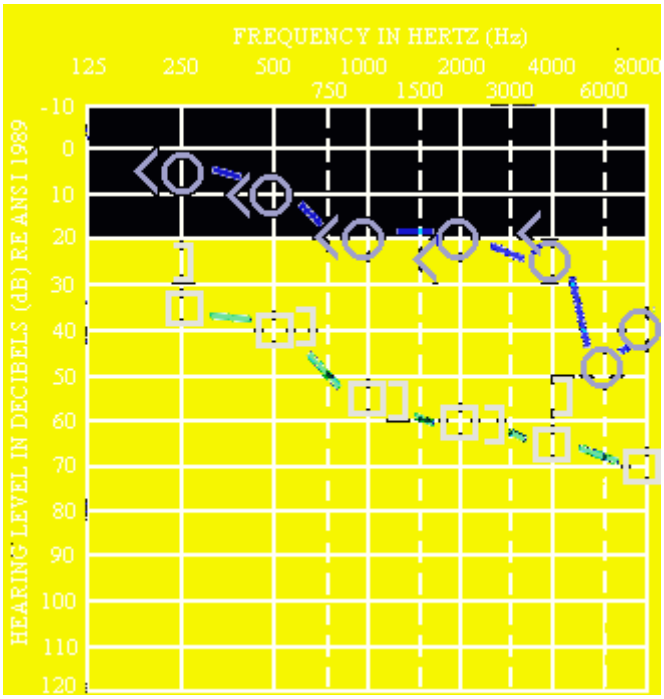
Test the better ear first. The tester should instruct the subject that the reason of the test is to detect the minimal sound that the subject is able to hear at several frequencies. The test should begin with a 1000 Hz sound and followed by 2000, 4000, 8000 and again 1000, then 500 and lastly 250 Hz.

The examiner first familiarizes the sound to the subject by presenting the sounds of supra threshold level (i.e. at 30dB). The pure tones should be delivered in 1 – 3 seconds in the interval of 3 seconds in between the sounds. The exact correct threshold is obtained after the tester gets at least 3 out of 5 responses correct. The subject responds by raising the finger or pressing the button of a light of audiometric panel. If the subject hears the particular tone, then the sound is decreased by 10dB and if he didn't hear, then the sound is increased by 5dB until he hears the sound. Similarly the second ear should be tested. This method is called as 5-up 10-down method[61].

# NORMAL PURETONE AUDIOGRAM




# HIGH FREQUENCY SENSORINEURAL HEARING LOSS



*Bone conduction threshold test*

Place the Bone conduction vibrator either over the mastoid bone or over the frontal bone with the help of the head band. Here the stimulus is produced by the vibrator and the tests are same like the air conduction test. The important difference is the test starts with 1000Hz, followed by 2000, 4000 and again 1000, 500 and 250Hz (8000Hz is not used here).

*Interpretation*

The values obtained from the air and bone conduction threshold tests are plotted in a pure tone audiogram. Intensity of sound in dB is plotted on the ordinate and the frequency in Hz is plotted on the abscissa. 'O' and 'X' symbols are used for right and left ear air conduction thresholds and < and > symbols are used for bone conduction thresholds of right and left ear respectively[61]. Red color is used for marking right ear data and Blue color is used for marking left ear data.

The purpose of puretone audiometry detects the type and degree of hearing loss. The degree of the hearing loss is assessed by calculating the average thresholds of 500 Hz, 1000 Hz and 2000Hz frequencies. These three frequencies are otherwise called as speech frequencies.

***Uses of pure tone audiometry***

1. Detecting the presence or absence of hearing impairment.
2. Identifying the degree of hearing loss and differentiating the sensorineural hearing loss from conductive loss.
3. Identifying the non organic component.
4. Used to predict the speech reception threshold.
5. Helps for hearing aid prescription.

***Classification of grades of severity of hearing loss***[62]

According to the American Speech Language and Hearing Association (ASHA),

| **Classification** | **Hearing level (dB)** |
|---|---|
| Normal hearing | - 10 to 15 |
| Minimal hearing loss | 16 to 25 |
| Mild hearing loss | 26 to 40 |
| Moderate hearing loss | 41 to 55 |
| Moderately severe hearing loss | 56 to 70 |
| Severe hearing loss | 71 to 90 |
| Profound hearing loss | >90 |

## Otoacoustic emissions

### *History*

Whenever external sounds stimulate the inner ear, the cochlea produce sounds responding to the stimulus. It occurs in all animals, even in dinosaurs. Till 1977, nobody suspected this fact. In 1940 George Von Bekesy demonstrated the sound travel as a wave on the basilar membrane. In 1948, Thomas Gold demonstrated that the cochlea causes the amplification of sounds.

In 1958, Elliot and Van Den Brink found that some specific 'ripples' limited the accuracy of the audiometric recordings. Later Flattorp and ward reported that some mysterious sound heard in their ears when tones applied externally in some frequencies.

In 1977, Van Bekesy and his friends demonstrated the cochlear echo produced in cochlea. In 1978, David Kemp described about the OAEs and after that Rudolph chum constructed the cochlear sounder based on Kemp's description[63]. It is a portable TEOAE (Transient Evoked OtoAcoustic Emission) instrument and demonstrated the click evoked acoustic response experiment.

In 1985, Alfred peter's limited company designed a first commercially prepared TEOAE instrument called Peter's AP200. In 1987, Siobhan Ryon designed a instrument ILO88 especially for the newborns with a screen called ILO88 newborn screen and he discovered that there should be reduction in 20dB in the stimulus probe, because of the small size of the newborn's ear canal.

In 1978, Kemp reported that OAEs were absent in the hearing loss patients. Stewert Anderson combined with Kemp confirmed the relationship between hearing and OAEs[64]. Duck On Kim was the first to observe the DPOAE (Distortion Product Oto Acoustic Emission) suppression is due to electrical stimulation of the efferent system of cochlea. In 1984 at London conference the clinical applications of DPOAE using a swept frequency DP tracking analyzer was demonstrated.

Otoacoustic emissions are the sounds produced by the outer hair cells of a normal cochlea and it travels through the middle ear and come to the ear canal and they can be recorded and measured by using a microphone. Thus the OAEs travel in reverse direction, i.e. from the cochlea to external ear canal.

OAEs are absent in the damaged cochlea and so it is used to test the cochlear functions. In eighth cranial nerve pathology the OAEs do not disappear, because the cochlear cells are normal. So OAE tests are used to differentiate cochlear and retro cochlear pathology.

***Classification of OAEs***

There are two types.

1. Spontaneous OAEs
2. Evoked OAEs
    a. Transient Evoked OAEs
    b. Distortion Product OAEs.

***Spontaneous OAEs (SOAEs)***

They are recorded without any external stimulation. They are present only in normal, healthy hearing persons. The hearing loss should not exceed 30dB in that person. There may be presence of 50% (40 to 70 %) of OAEs in normal individuals and may be absent in remaining persons.

***Transient Evoked OAEs (TEOAEs)***

TEOAEs are measured after the presentation of a click or tone burst into the ear. The response occurs after a delay of 5ms. A series of 80 to 85dB clicks are used to evoke the response. The response consists of two waveforms. Comparison of these waveforms gives the noise level in dB of SPL. TEOAEs were nearly absent in all sensorineural hearing loss ears.

**Distortion Product Oto Acoustic Emissions (DPOAE)**

These are the evoked response Oto Acoustic Emissions (Distortion products) which are produced by introducing two tones with different frequency simultaneously into the cochlea. The cochlear echo detected after 5ms after a click, peaks at latencies of approximately 5 to 15ms. This phenomenon is called as Kemp echo[15].

DPOAEs are used to analyze the range of hearing from 1000 to 8000Hz. DPOAE threshold used to detect the presence of severe or total hair cell damage with good specificity[65]. DPOAE amplitude reduced in proportion to the amount of OHC loss. So the DPOAEs study reflects the functional integrity of the OHCs[66, 67].

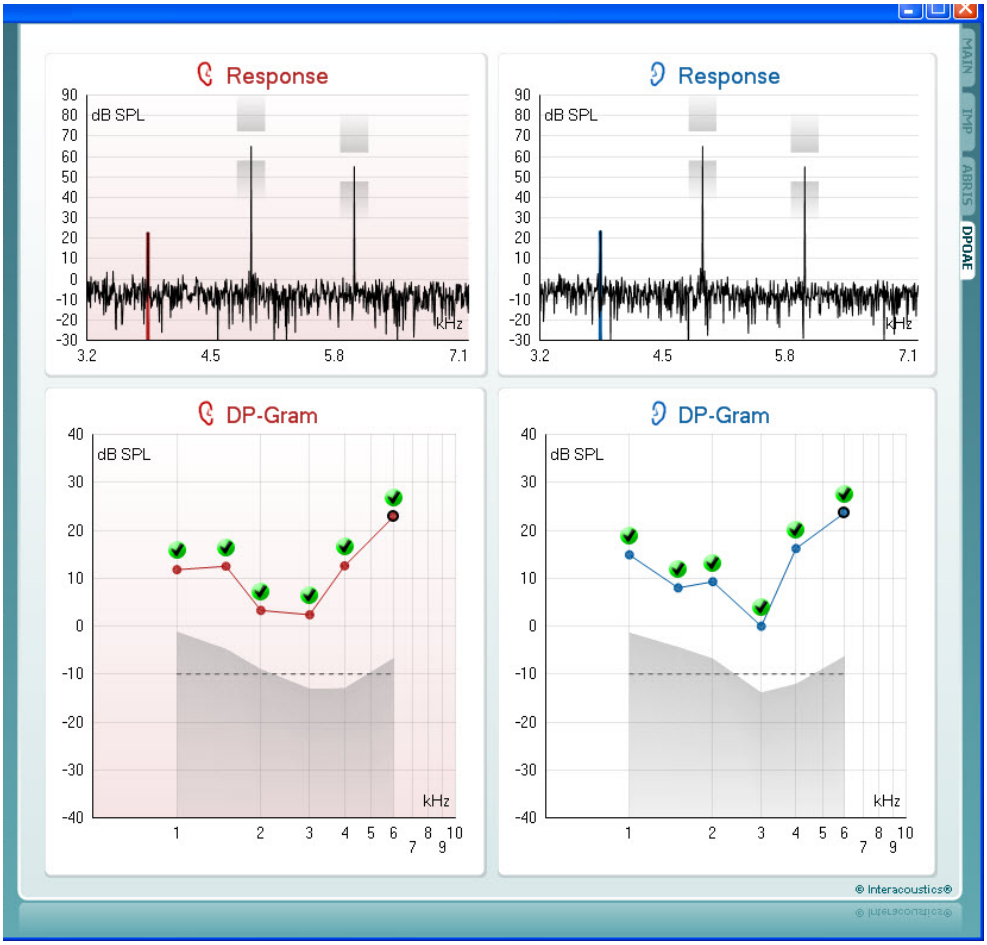
***Procedure and interpretation***

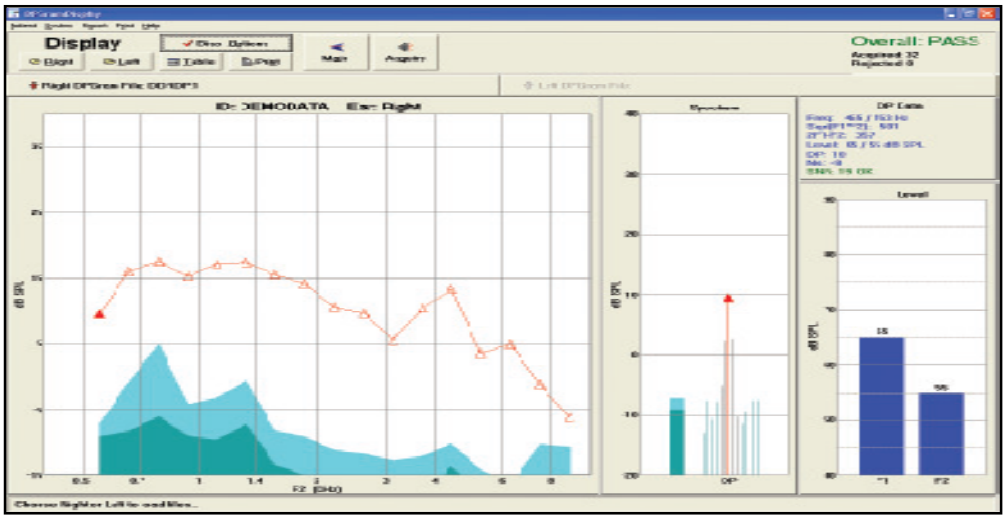
It is a non invasive test and doesn't require any sedation of the persons. It takes only 2 to 5 minutes for both the ears and specialized sound proof room is not necessary, but a quiet room is enough.

Grason Staddler (GS I) device was used to study the DPOAE recordings. The instrument contains a probe and a computer software called Intelligent Hearing Systems. Microphone-Stimulator probes like the 10D, are self contained units that can deliver the stimulus and pick up the response.

Two clicks or pure tones called 'primaries' are sent through a probe which is inserted into the external auditory meatus. The clicks are generated by the acoustic stimulus and the resulting DPOAEs, which are produced by the cochlea are picked up by a microphone. The emissions are digitalized and analyzed by the computer. The frequencies used to generate the stimulus are from 500Hz to 8000Hz. The system will analyze the responses to each sweep and plot them in a DPGram, enhancing response analysis.

# DPOAE RECORDINGS – DP GRAM



A DPGram will display the intensity of the response to each frequency point and the noise level. The DPOAE system also displays passing criteria for each frequency point.

***Mechanism of DPOAE***

The emission and stimulus will interact with each other and result in peaks and valleys at frequencies. The frequencies of the tones are designated as $f_1$ and $f_2$. These two frequencies are close in frequency and both interact with basilar membrane results in energy output of cochlea at discrete frequencies that are related to the tone frequencies ( e.g. f2-f1, 2f1-f2, 3f1-2f2, 2f2-f1 )[68]. The correlations with hearing thresholds were measured better with the DP-grams of the nonlinear component of the $2f2 - f1$ DPOAEs[69].

DPOAEs are measured by narrow band filtering at the particular frequencies. DPOAEs are the combination of energy from the reflection component of DPOAE frequency and a non linear distortion component of the two tones. These effects are seen only at the lower level of the stimulus and it is disappearing when the stimulus level more than 40 to 50 dB level.

***Uses of OAEs***

1. It helps in assessing the hearing in neonates and also in mentally retarded persons or uncooperative patients.
2. OAEs are helping in diagnosing retro cochlear pathology.
3. They used to distinguish retro-cochlear from cochlear hearing loss.
4. Ototoxic effects are detected earlier before detected by pure tone audiometry.
5. They identify the malingering persons and non pathological hearing loss.
6. OAEs are not affected by Sedation.

**Studies of hearing impairments in alcoholics**

A case control study conducted by Kavitha Ashok Kumar in hearing in alcoholics revealed that alcohol consumption is the single risk factor for deafness[52]. And also found that mild to moderate hearing loss present in the alcoholics.

Juen Haur Hwang et al, in their study of the acute effects of alcohol on auditory thresholds and distortion product otoacoustic emissions in humans, found that DPOAE amplitudes were reduced at high frequencies in humans who consumed moderate amounts of alcohol.

They suggest that alcohol affected hearing via the nervous system and also affecting the functions of OHCs[51].

In the study of alcoholism: the effect on the cochleo vestibular apparatus, Marcieli Belle et al, reported that 55% of alcoholics with age group between 33 and 49 had a hearing impairment whereas 82.35% of alcoholics with age group between 50 and 70 had hearing impairment. They concluded that alcohol interferes both hearing and also individual's balance[70].

Long term alcohol abuse causes ototoxity and it affects the cochlear function, especially the external hair cells. This was the conclusion of Sandra Beatriz et al study of auditory assessment of alcoholics in abstinence in 2005 with 75 alcoholics[11].

In the study of the acute effects on auditory thresholds, the author Tahwinder Upile et al from London university hospitals, suggested that alcohol mainly impaired the lower frequency thresholds which included 1000Hz which is the frequency to discriminate vowels. He also described that short term alcohol effects were reversible with treatment, but long term effects are irreversible[7].

Sharon G. Curhan et al in his article had the title of a prospective study of alcohol use and hearing loss in men observed that the consumption of white wine for 2 to 4 times a week causes increased risk of hearing loss[9]. And they also suggest that higher risk of hearing loss in alcoholics with lower intake of supplements of vitamin B12. Alcohol causes vitamin B12 deficiency which is important in myelin synthesis, cellular metabolism in cochlear organs also. They present few evidences that alcohol may help preserve hearing through optimal cochlear blood flow[9].

Niedzieiska G, et al in their study of hearing loss in chronic alcoholics told that chronic excessive drinking of alcohol causes hearing loss, sight deterioration and some psychical disorders. They suggested that the sensorineural hearing loss is due to the outer hair cells damage and also the hearing pathway damage of the brain stem. It causes the loss of otoacoustic emissions[71].

Merton LJ, et al demonstrated that alcohol significantly increased the dynamic range (C levels) of sound perception in cochlear implant users and this effect is due to the alteration in the auditory pathways proximal to the cochlea.

They described the cochlear effects of alcohol in their study of a prospective randomized controlled trial evaluating alcohol on loudness perception in cochlear implant user[72].

In the study of types of hearing disorders in drug addicts and individuals drinking non consumable alcohols, Lukomski M, et al found that alcoholics had a hearing impairment and tinnitus and the addicts have more percentage of hearing impairment among 210 subjects[73].

A Russian study of effect of alcoholic intoxication on various indicators of the functional state of the acoustic analyzer by Guliamov MG, showed alcohol causes more disorders of central parts of the auditory system. Chronic alcoholic intoxication causes the steady dysfunction in the form of treble hypoacusis in cochlea[74].

Robinette MS et al, in their study of the effects of alcohol on the acoustic Reflex Relaxation Index, discussed that there was an abnormality in the effect of specific temporal stimulus pattern in the Reflex Relaxation Index of the alcoholics. They also concluded that alcohol produces sensorineural hearing impairment[75].

Golabek W and Niedzielska G noted that 70% of alcoholics had sensorineural hearing loss and the hearing loss is associated with the duration of heavy drinking. He also noted that the results of their tests indicated that in chronic alcoholics the lesion is mainly in retro-cochlear areas of inner ear[76].

German article of Vitamin A concentration in plasma and ability to hear in patients with chronic alcoholic liver diseases, the author Lohle E, and Scholmerich J et al found that the frequencies from 2000 Hz causes depression of the pure tone threshold in the chronic alcoholic patients[10]. The abnormality in the Acoustic facial reflex test and the Carhart-test indicated that the cochlear lesions were present in 50% of alcoholics. They also showed that alcohol with deficiency of vitamin A leads to impaired hearing[10].

The auditory system transmission breakdown in alcoholics was proved by Spitzer JB in his study of auditory effects of chronic alcoholism. He demonstrated that the impairment by many techniques like histologic evidence, evoked potential data, threshold measurement, behavioral central auditory evaluation and acoustic reflex studies[77].

Wheeler DC showed a bilateral high frequency loss consistently in 52 alcoholic subjects and he found that the loss is related to the duration of time, but not dependent on age in his study of Audiometric configuration in patients being treated for alcoholism[78].

Beam SL observed impairments in hearing, language, speech, and voice in 14 out of 15 alcoholics in treatment, in the study of Communication deviations in alcoholics; a pilot study[79].

In the March 2004 issue of Alcoholism: Clinical & Experimental Research, scientists in Germany specified that the damage that cumulative, lifelong alcohol consumption can inflict on central auditory pathways, which is reflected as hearing loss[54].

A study of 20 long term alcohol dependence patients on Audiovestibular dysfunction by Roshan K. Verma, Naresh K. Panda and they demonstrated that the hearing threshold levels of alcohol dependence subjects were elevated at 4000 and 8000Hz[80].

In the study of Effects of alcohol on the outer hair cell protein Prestin (775.2). Prestin is a transmembrane motor protein, which is present in the outer hair cells.

For cochlear amplification and electro motility, outer hair cells need the motor protein Prestin which depends on cholesterol level of cell membrane[37]. Elizabeth Minten studied that alcohol modulate the lipid bilayer properties of cell membrane and thereby affect the amplification of outer hair cells which leads to sensorineural hearing loss [37].

Jozsef Nagy showed that chronic alcohol consumption causes increased levels of NMDA receptor coagonist homocysteine and caused apoptotic neurotoxicity and brain atrophy[56].

The apoptotic neurotoxicity caused the loss of hair cells and supporting cells which lead to hearing loss. They also explained the alteration in the neurotransmitter levels in chronic alcoholism in their study of Alcohol related changes in regulation of NMDA receptor Functions.

In the study of Disability associated with Alcohol abuse and dependence, Andriy V. Samokhvalov et al showed that the supplementary attribute of alcohol were anxiety, impairment in hearing and speech[49].

Phillip M. Uribel et al in their larval study of Ethanol affects the development of Sensory hair cells in Larval Zebrafish, they showed that increased hair cell death and decreased proliferation of supporting cells by treating with ethyl alcohol caused the damaged to the auditory apparatus[53].

Alcohol had a toxic effect on cochlear and vestibular function by reduce the Auditory Brainstem Response which was demonstrated by Perez R and Freeman S et al in their study of Vestibular and cochlear ototoxicity of topical antiseptics assessed by evoked potentials in the sand rat.[50]

# *MATERIALS & METHODS*

# MATERIALS AND METHODS

## Study design

This is a cross sectional study.

## Study place

The study was conducted in the Department of Physiology, Coimbatore Medical College, Coimbatore.

## Collaborating department

Department of Otorhinolaryngology and Department of Psychiatry, Coimbatore Medical College Hospital, Coimbatore.

## Study period

The study was conducted from August 2013 to June 2014. Before the start of the study, the approval was obtained from the ethical committee of the Coimbatore Medical College Hospital.

## Study subjects

A total of 134 subjects (males) in the age group of 25 to 55 years were included in the study and divided into control and study group, 67 in each group.

**The control group**

67 apparently healthy males who have never consumed alcohol were included in the control group (college students, office staffs, businessmen and coolies).

**The study group**

The study subjects were alcoholics of more than 2 years duration were selected from the outpatient department of Psychiatry, Coimbatore medical college, Coimbatore who were coming for deaddiction therapy. They were selected by using AUDIT questionnaire.

Pure tone audiometry was recorded in the department of Physiology. The study group (alcoholics) consists of business men, office staffs and coolies working in a noiseless surroundings. The alcoholics should be well oriented for the procedures and the surrounding environment. The DPOAE recordings were done in the Department of Otorhinolaringology.

**Exclusion criteria**

Patients who were above 60 years of age were excluded. The subjects have diabetes mellitus, hypertension, ear infections, any congenital anomalies of ear and those on treatment with ototoxic drugs were excluded. The workers with the history of occupational exposure to noise were excluded from the study.

# AUDIT QUESTIONNAIRE

**Box 10**

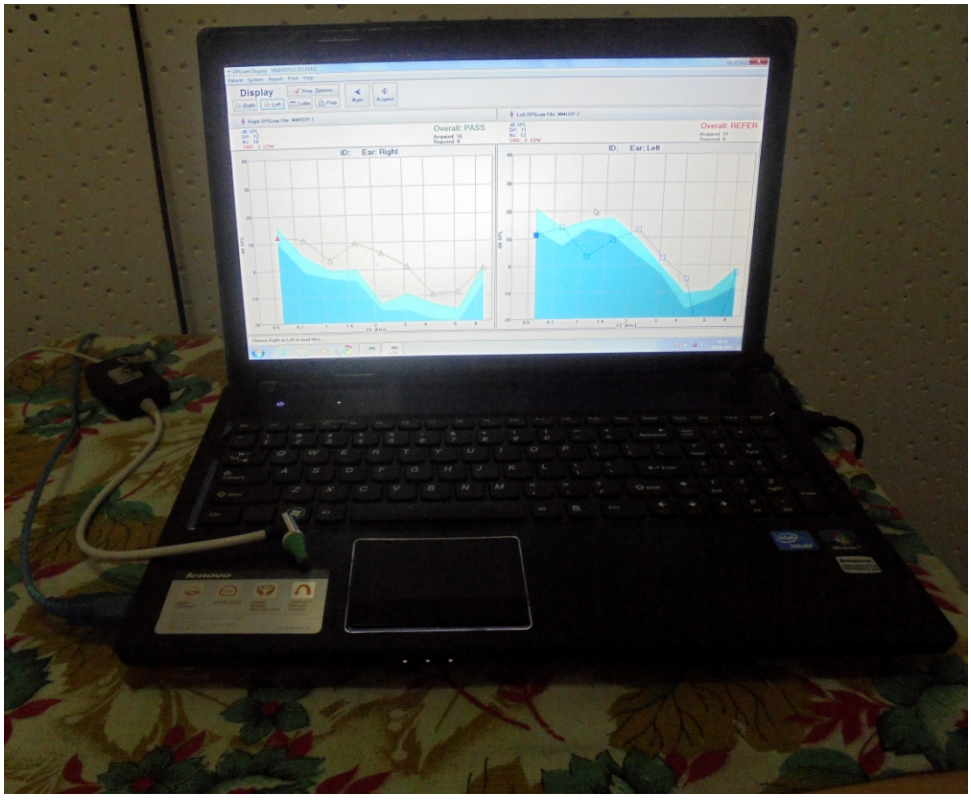
## The Alcohol Use Disorders Identification Test: Self-Report Version

PATIENT: Because alcohol use can affect your health and can interfere with certain medications and treatments, it is important that we ask some questions about your use of alcohol. Your answers will remain confidential so please be honest.
Place an X in one box that best describes your answer to each question.

| Questions | 0 | 1 | 2 | 3 | 4 | |
|---|---|---|---|---|---|---|
| 1. How often do you have a drink containing alcohol? | Never | Monthly or less | 2-4 times a month | 2-3 times a week | 4 or more times a week | |
| 2. How many drinks containing alcohol do you have on a typical day when you are drinking? | 1 or 2 | 3 or 4 | 5 or 6 | 7 to 9 | 10 or more | |
| 3. How often do you have six or more drinks on one occasion? | Never | Less than monthly | Monthly | Weekly | Daily or almost daily | |
| 4. How often during the last year have you found that you were not able to stop drinking once you had started? | Never | Less than monthly | Monthly | Weekly | Daily or almost daily | |
| 5. How often during the last year have you failed to do what was normally expected of you because of drinking? | Never | Less than monthly | Monthly | Weekly | Daily or almost daily | |
| 6. How often during the last year have you needed a first drink in the morning to get yourself going after a heavy drinking session? | Never | Less than monthly | Monthly | Weekly | Daily or almost daily | |
| 7. How often during the last year have you had a feeling of guilt or remorse after drinking? | Never | Less than monthly | Monthly | Weekly | Daily or almost daily | |
| 8. How often during the last year have you been unable to remember what happened the night before because of your drinking? | Never | Less than monthly | Monthly | Weekly | Daily or almost daily | |
| 9. Have you or someone else been injured because of your drinking? | No | | Yes, but not in the last year | | Yes, during the last year | |
| 10. Has a relative, friend, doctor, or other health care worker been concerned about your drinking or suggested you cut down? | No | | Yes, but not in the last year | | Yes, during the last year | |
| | | | | | **Total** | |

## PURE TONE AUDIOMETER


## SMART DPOAE



**The materials used for the study**

*AUDIT questionnaire*[81] – To select the alcoholics by the set of questions.

*The Proforma* - To enter the details of cases and controls and to record the clinical findings on examination.

*Puretone audiometer* - To record the conventional audiometry findings. MAICO- MA 52 with Telephonics TDH 39p supra aural earphones and Bone conduction vibrator B71 was used.

*Grason Staddler (GS I) device* - To record the DPOAE findings.

**Methodology**

The study was initiated after clearly explaining the procedures in detail. The informed written consent was obtained from the subjects. The study proforma contains,

1. Name, age, sex, occupation and history of diabetes mellitus, hypertension, and family history of any congenital anomalies of ear.
2. Alcoholic history - type of alcohol, consumption of alcohol in units and duration of alcoholic beverages on a regular basis.

# RINNE'S TEST

**Otoscopic examination**

The examination of the ear was done with the otoscopy by the Otorhinolaringologist. It included the examination for the occurrence of any infections of the ear like otitis media, otorrhoea, ear wax, any foreign body, any abnormality in the external auditory meatus, bleeding and injury or abnormality of the tympanic membrane.

**Tuning fork tests**

*Rinne's test* :- To compare the air and bone conduction, Rinne's test was carried out in each ear separately, in both the controls and cases. The test was done by placing the foot plate of the vibrating tuning fork on the mastoid process and asking the subject to raise the hand whenever he stops hearing. Immediately vibrating tuning fork was placed in front of the meatus and ask him whether he hears the sound. In the normal subjects air conduction is better than bone conduction (Rinne positive) and also in sensorineural hearing loss. Bone conduction is better than air conduction (Rinne negative) in conductive deafness. The test was then repeated on the other ear.

# WEBER'S TEST

*Weber's test*:- The vibrating tuning fork is placed in the centre of the forehead and the subject is asked to indicate whether he hears the sound equally in both ears or hears better in one ear. The normal response is to hear the sound in the midline. If the sound is heard best in the defective ear, the hearing loss is due to conduction deafness. If the sound is heard louder in the normal ear, the hearing loss is due to sensorineural deafness[82].

**Procedure for recording puretone Audiometry**

In both the cases and controls the audiometry was done in the sound proof room at the department of physiology, Coimbatore medical college, Coimbatore. According to the recommendations of the American Speech language and Hearing Association, the calibration of the MAICO MA 52 clinical digital audiometer was checked and the audiometric testing was conducted.

The subjects were explained clearly that, they should respond even to mild sound, by raising their fingers when the pure tone is heard. The test needed full co-operation of the person because, it is a subjective test. The test was first performed in the better ear. The diaphragm of the supra aural earphones must be placed correctly over the auditory meatus.

# RECORDING OF PURE TONE AUDIOMETRY

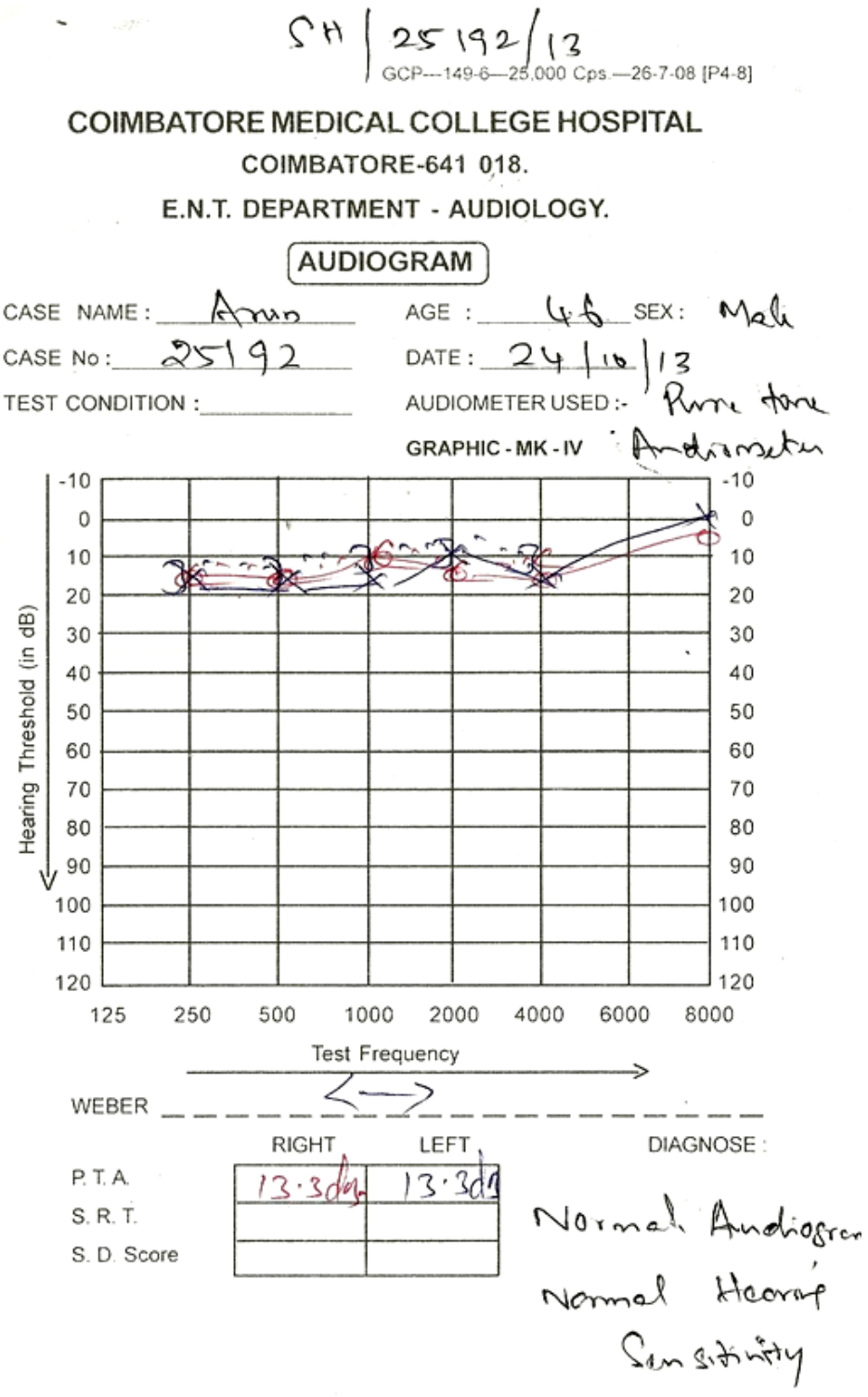
Better ear is tested initially at 1000Hz for one to three seconds duration. The greatest test–retest reliability was more at 1000Hz that is why we were using this frequency. The procedure was initiated by presenting the supra threshold intensity of 30dB for normal hearing people and more than 30dB above the estimated threshold for the subject with hearing impairment, so as to familiarize the tone to the subjects. But the initiating intensity should never go above 80dB.

If the tone is not heard then the intensity was increased by 5dB. Likewise, increasing 5dB steps till he hears the sound. Once he hears the sound, then the intensity is reduced by steps of 10dB till he fails to give a positive response.

The tone was increased by 5dB and decreased by 10dB till the subject responds correctly for at least 3 out of 5 responses at the same level that being hearing threshold level of that subject. Similarly the next frequency 2000dB was tested by starting at the threshold level by using the 5-up 10-down procedure.

All the frequencies were recorded in the order of 1000, 2000, 4000, 8000 and again 1000Hz then 500 and finally 250Hz. Similarly the second ear also tested by starting with the last frequency used for testing the first ear.

**Audiogram of a control showing normal hearing sensitivity**



SH | 25192/13

GCP---149-6—25,000 Cps.—26-7-08 [P4-8]

COIMBATORE MEDICAL COLLEGE HOSPITAL

COIMBATORE-641 018.

E.N.T. DEPARTMENT - AUDIOLOGY.

AUDIOGRAM

CASE NAME : Arun AGE : 45 SEX : Male

CASE No : 25192 DATE : 24/10/13

TEST CONDITION : AUDIOMETER USED :- Pure tone Audiometer

GRAPHIC - MK - IV

Hearing Threshold (in dB)

-10 0 10 20 30 40 50 60 70 80 90 100 110 120

125 250 500 1000 2000 4000 6000 8000

Test Frequency

WEBER

| | RIGHT | LEFT |
|---|---|---|
| P.T.A. | 13.3 | 13.3 |
| S.R.T. | | |
| S.D. Score | | |

DIAGNOSE :

Normal Audiogram

Normal Hearing Sensitivity

**Audiogram of a case showing minimal hearing loss**

SH/ 22212/13

GCP---149-6---25.000 Cps.---26-7-08 [P4-8]

COIMBATORE MEDICAL COLLEGE HOSPITAL

COIMBATORE-641 018.

E.N.T. DEPARTMENT - AUDIOLOGY.

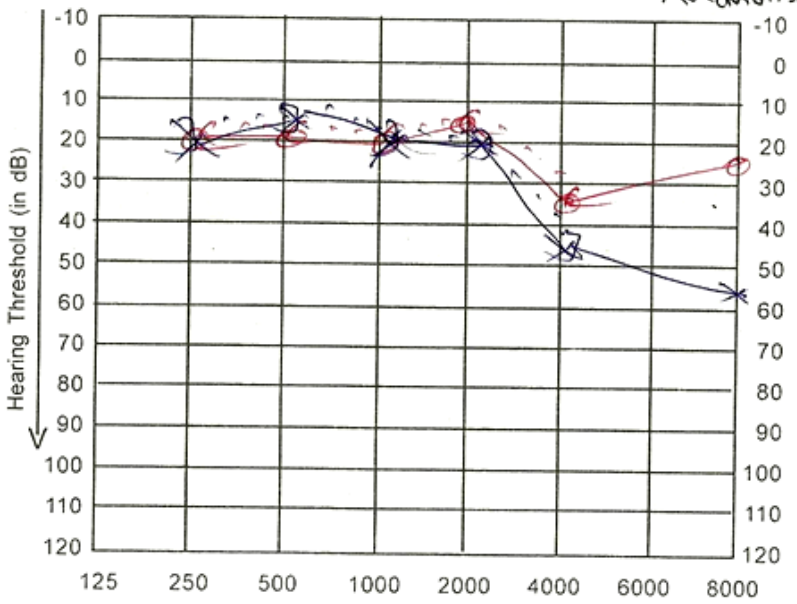
AUDIOGRAM

CASE NAME : Muniappan AGE : 55 SEX : Male

CASE No : 22212 DATE : 4/9/13.

TEST CONDITION : AUDIOMETER USED :- Pure tone Audiometer

GRAPHIC - MK - IV




WEBER

| | RIGHT | LEFT |
|---|---|---|
| P. T. A. | 18.3dB | 18.3dB. |
| S. R. T. | | |
| S. D. Score | | |

DIAGNOSE : B/L Minimal Sensorineural loss ↑ 4kHz & 8kHz dip

The American Speech Language and Hearing Association recommended symbols used in the audiogram are

| | Right ear | Left ear |
|---|---|---|
| Air conduction | O | X |
| Bone conduction | < | > |
| No response |  |  |

The red color symbol used in the audiogram indicates the right ear and blue color indicate the left ear. The connecting lines used for indicating air conduction are continuous line, whereas broken line indicates the bone conduction.

## Distortion product otoacoustic emission test

DPOAE recordings were done in the Department of Otorhinolaryngology, Coimbatore Medical College Hospital, Coimbatore uses Grason Staddler (GS I) device (smart DPOAE) with Intelligent hearing system software.

# RECORDING OF DPOAE

**Patient Preparation**

The subject is placed in a comfortable and quiet environment. The procedure was explained clearly to the subjects. The ear should be free from any wax or any materials and is confirmed by otoscopy. Microphone-Stimulator probes, used with an appropriate tip to provide a good seal, to reduce external noise.

**Setting up smart DPOAE**

After entering the parameters, number of sweeps, block size, intensity levels, artifact level allowed and number of retries are set on the General Tab. On the Frequency Tab, the start and end frequencies, number of frequencies per octave and F2/F1 ratio are set. The right button is pressed to acquire DPOAE's for the right ear. Then the probe is changed to the left ear and the left button is pressed.

The recommended settings for diagnostic DPOAE acquisition are,

• ***Frequencies :*** 500 – 8000 Hz.

• ***Frequencies per Octave*** **:** 3

• ***F2/F1 ratio*** **:** 1.22

• ***Sweeps :*** 16 sweeps per frequency.

• ***Block Size :*** 8 sweeps per block.

• ***Leve l:*** 65dB SPL for Level 1 and 55dB SPL for Level 2

• ***Passing Criteria :*** 70% or higher**.**

**Analysis**

As the test is being completed, the system averages the responses to each sweep and place them in a DP Gram, facilitating response analysis. A DP Gram will display the intensity of the response at each frequency point and the noise level around it. The frequencies of the tones are designated as f1 and f2. The DPOAEs measured by narrow band filtering of $2f_1$-$f_2$ at that particular frequency. The special pressure level was calculated and finally the results obtained as "pass" or "refer" criteria [83]. 70% response or more than 70% were taken as the correct response and indicated as 'Pass' and less than 70% responses were indicated as "Refer". Refer response is due to the absence of emissions from the cochlea and it indicates that some damage occurred to the hair cells of the cochlea.

The common finding for most of the alcoholics was increased threshold for higher frequency sounds like 4000Hz and 6000Hz.

# HIGH FREQUENCY LOSS DP-OAE RECORDING –

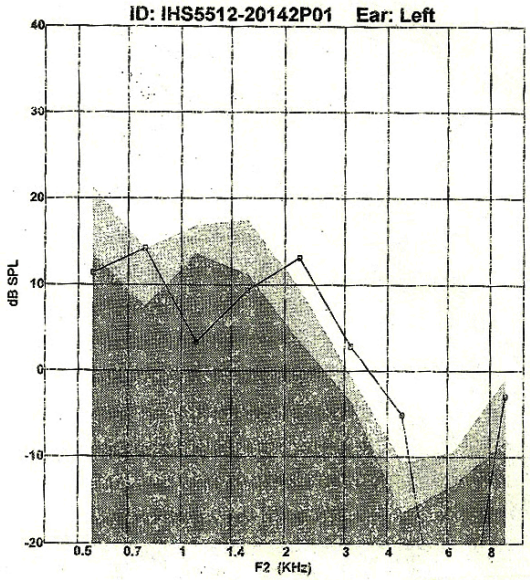
# REFER CRITERIA

SmartEP ASSR OAE VRA
INTELLIGENT HEARING SYSTEMS
6860 SW 81st Street Miami, Florida 33143
305-668-6102 • 1-800-IHSYSTEMS • www.ihsys.com

**SmartOAE 5.00** **DP-OAE Report**

COIMATORE MEDICAL COLLEGE AND HOSPITAL
1590/1-12, Trichy Road
COIMBATORE, TAMILNADU 641018
0422-2301393

Name: muniappan m
ID#:
DOB: 1/1/1969
Sex: Male




MMLDP.2

REFER

Date: Feb 25, 2014, 9:01:17 AM
Ear: Left Frqs: 9 File: C:\...\IHS5512-2014\MMLDP.2

| Frequencies: (Hz) | | | Swps | Amplitudes: (dB SPL) | | | | | | | SNR |
|---|---|---|---|---|---|---|---|---|---|---|---|
| F1 | F2 | Fdp | | L1 | L2 | A1 | A2 | DP | Ns | SNR | Stat |
| 455 | 553 | 357 | 16 | 65 | 55 | 65 | 54 | 11 | 13 | -2 | LOW |
| 641 | 783 | 499 | 16 | 65 | 55 | 65 | 54 | 14 | 7 | 7 | OK |
| 905 | 1105 | 704 | 16 | 65 | 55 | 66 | 54 | 3 | 14 | -10 | LOW |
| 1281 | 1560 | 1003 | 16 | 65 | 55 | 65 | 54 | 9 | 11 | -2 | LOW |
| 1810 | 2211 | 1409 | 16 | 65 | 55 | 64 | 55 | 13 | 3 | 10 | OK |
| 2563 | 3125 | 2000 | 16 | 65 | 55 | 65 | 55 | 3 | -4 | 7 | OK |
| 3619 | 4416 | 2822 | 16 | 65 | 55 | 65 | 55 | -5 | -17 | 11 | LOW |
| 5120 | 6250 | 3991 | 16 | 65 | 55 | 65 | 55 | -41 | -13 | -27 | LOW |
| 7243 | 8837 | 5649 | 16 | 65 | 55 | 66 | 57 | -3 | -8 | 5 | LOW |

# NORMAL DP-OAE RECORDING – PASS CRITERIA

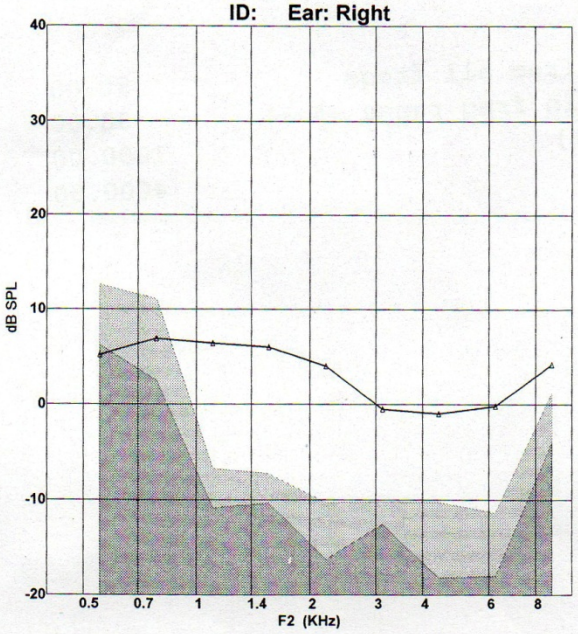
INTELLIGENT HEARING SYSTEMS

6860 SW 81st Street Miami, Florida 33143
305-668-6102 • 1-800-IHSYSTEMS • www.ihsys.com

**SmartOAE 5.00** **DP-OAE Report**

COIMATORE MEDICAL COLLEGE AND HOSPITAL
1590/1-12, Trichy Road
COIMBATORE, TAMILNADU 641018
0422-2301393

Name: govindaray k
ID#:
DOB: 6/10/1977
Sex: Male




GKRDP.1

PASS

Date: Feb 17, 2014, 9:09:32 AM
Ear: Right Frqs: 9 File: C:\...\IHS5512-2014\GKRDP.1

| Frequencies: (Hz) | | | Swps | Amplitudes: (dB SPL) | | | | | | | SNR |
|---|---|---|---|---|---|---|---|---|---|---|---|
| F1 | F2 | Fdp | | L1 | L2 | A1 | A2 | DP | Ns | SNR | Stat |
| 455 | 553 | 357 | 16 | 65 | 55 | 63 | 53 | 5 | 6 | -0 | LOW |
| 641 | 783 | 499 | 16 | 65 | 55 | 63 | 53 | 7 | 2 | 4 | LOW |
| 905 | 1105 | 704 | 16 | 65 | 55 | 63 | 54 | 6 | -11 | 17 | OK |
| 1281 | 1560 | 1003 | 16 | 65 | 55 | 66 | 55 | 6 | -10 | 16 | OK |
| 1810 | 2211 | 1409 | 16 | 65 | 55 | 66 | 53 | 4 | -16 | 20 | OK |
| 2563 | 3125 | 2000 | 16 | 65 | 55 | 60 | 49 | -0 | -13 | 12 | OK |
| 3619 | 4416 | 2822 | 16 | 65 | 55 | 65 | 54 | -0 | -18 | 17 | OK |
| 5121 | 6250 | 3991 | 16 | 65 | 55 | 64 | 55 | -0 | -18 | 18 | OK |
| 7243 | 8837 | 5649 | 16 | 65 | 55 | 66 | 53 | 4 | -4 | 8 | OK |

The hearing impairment is classified according to the WHO classification of hearing loss. The affected alcoholics have minimal to mild hearing loss (Minimal hearing loss = 16 to 25dB, Mild hearing loss = 26 to 40dB).

# *STATISTICAL ANALYSIS*

## STATISTICAL ANALYSIS

The audiometric findings and DPOAE recordings were collected and recorded in a Master Chart. The Data analysis was done with the help of computer using **Epidemiological Information Package (EPI 2010)** produced by Centre for Disease Control, Atlanta.

Using this software frequencies, range, percentages, standard deviations, means, 'p' values and chi square were calculated. Unpaired ‘t’ test was used to analyze the significance of difference between quantitative variables. For qualitative variables, Yate’s and Fisher’s chi square tests were used. A 'p' value less than 0.05 is taken to denote significant relationship between the variables.

# *RESULTS*

# RESULTS

The present study was conducted in the Department of Physiology, Otorhinolaryngology, and the Department of Psychiatry, Coimbatore Medical College Hospital, Coimbatore. A total of 67 healthy individual males and 67 alcoholic males were included in the study. The study subjects were divided into two groups based on their intake of alcohol.

Group A - 67 cases of alcoholics age matched males.

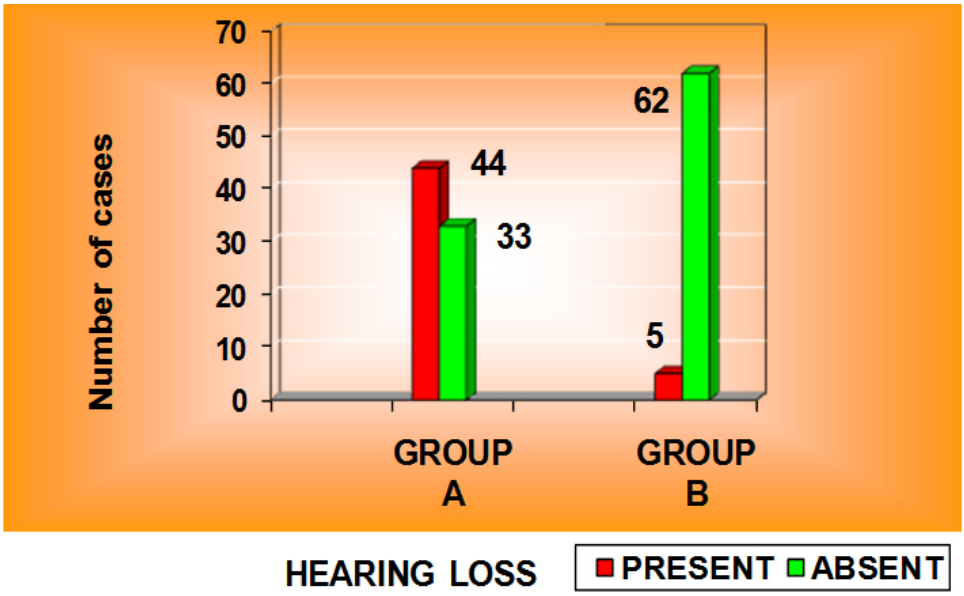
Group B - 67 controls, age matched males.

The hearing loss for both groups were assessed by pure tone audiometer and DPOAEs. It was found that alcohol causes sensorineural hearing loss at high frequency of more than 3000Hz.

**Table 1: Hearing Loss as per Pure Tone Audiometry in Cases and Controls**

| Group | Hearing Loss | | | |
|---|---|---|---|---|
| | Present | | Absent | |
| | No | % | No | % |
| Group A | 44 | 65.7 | 33 | 34.3 |
| Group B | 5 | 7.5 | 62 | 92.5 |
| 'p' | <0.0001 Significant | | | |

**Figure 1 : Hearing Loss (As Per PTA)**

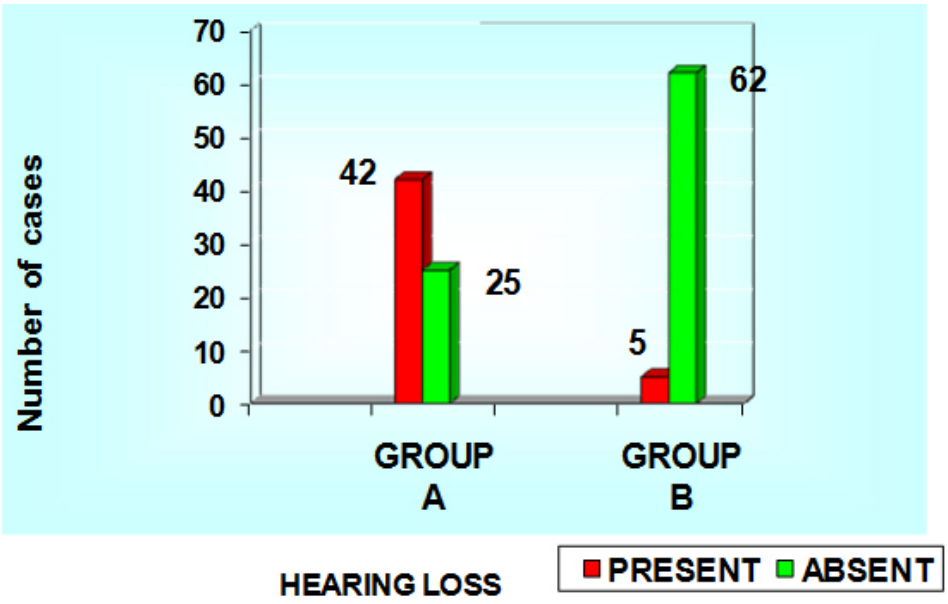



With puretone audiometry there is a significant (P<0.001) hearing loss among the alcoholics (65.7%) when compared with the Non-alcoholics (7.5%).

**Table 2 : Hearing Loss as per DPOAE recordings in Cases and Controls.**

| Group | Hearing Loss as per DPOAE | | | |
|---|---|---|---|---|
| | Present | | Absent | |
| | No | % | No | % |
| Group A | 42 | 62.7 | 25 | 37.3 |
| Group B | 5 | 7.5 | 62 | 92.5 |
| 'p' | <0.0001 Significant | | | |

**Figure 2 : Hearing Loss (As Per DPOAE)**

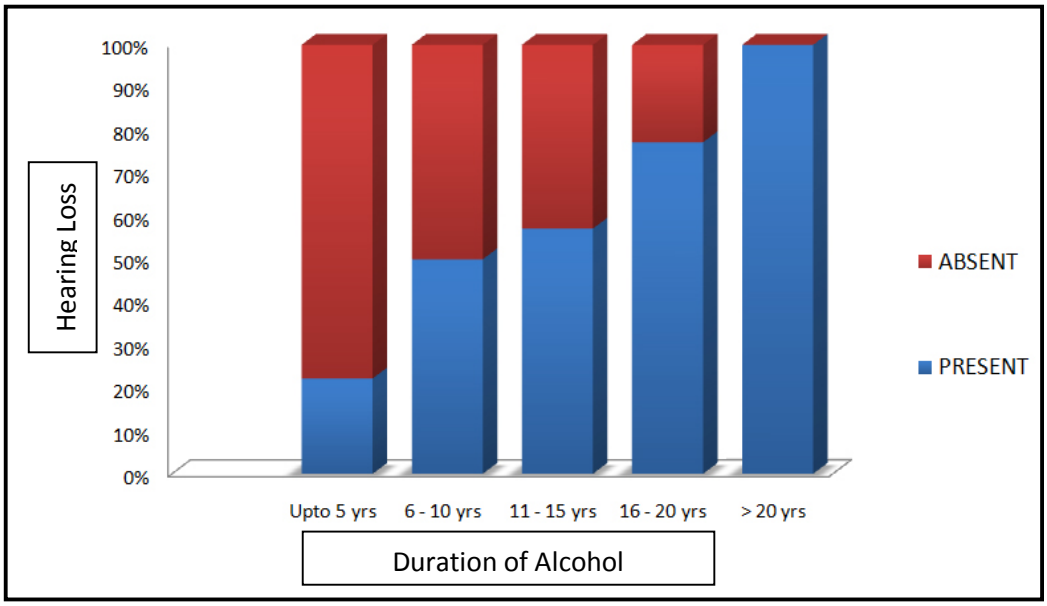



The DPOAE recordings also showed that there is a significant (P<0.001) hearing loss among the alcoholics (62.7%) when compared with the Non-alcoholics (7.5%).

**Table 3 : Effect of duration of alcoholism on hearing**

| Duration of Alcoholism | Number of Subjects | Hearing Loss | | | |
|---|---|---|---|---|---|
| | | Present | | Absent | |
| | | Mean | SD | Mean | SD |
| Up to 5 yrs | 9 | 2 | 22.2 | 7 | 77.8 |
| 6 – 10 yrs | 12 | 6 | 50 | 6 | 50 |
| 11 – 15 yrs | 21 | 12 | 57.1 | 9 | 42.9 |
| 16 – 20 yrs | 18 | 17 | 94.4 | 1 | 5.6 |
| >20 yrs | 7 | 7 | 100 | - | - |
| **Duration (years)** | | | | | |
| Mean | | 15.98 | | 9.09 | |
| SD | | 5.84 | | 4.22 | |
| **'p'** | | **<0.0001 Significant** | | | |

**Figure 3 : Duration of alcoholism & hearing loss among alcoholics**

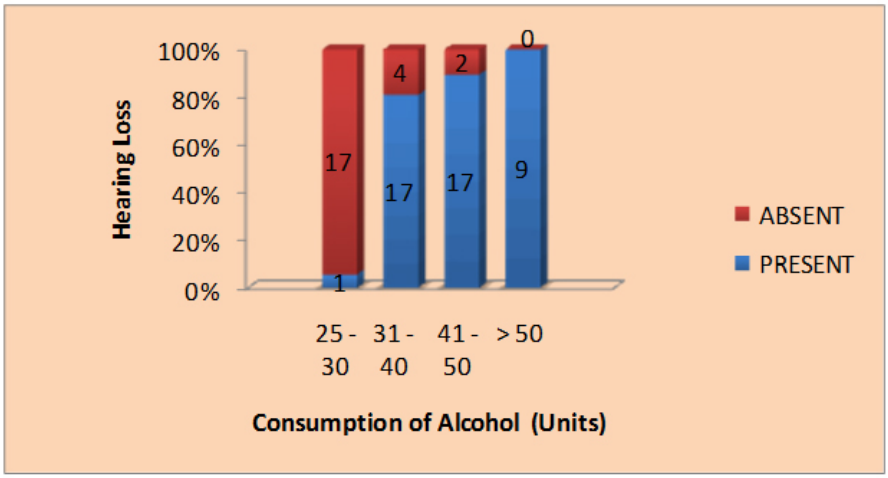



There is a significant (P<0.001) hearing loss among the alcoholics with regards to the duration of alcohol intake.

**Table 4 : Effect of amount of Consumption of alcohol on hearing.**

| Units of alcohol consumption per week | Number of Subjects | Hearing Loss | | | |
|---|---|---|---|---|---|
| | | Present | | Absent | |
| | | Mean | SD | Mean | SD |
| 25 – 30 | 18 | 1 | 5.6 | 17 | 94.4 |
| 31 – 40 | 21 | 17 | 81.0 | 4 | 19.0 |
| 41 – 50 | 9 | 17 | 89.5 | 2 | 10.5 |
| Above 50 Units | 19 | 9 | 100 | - | - |
| **Units** | | | | | |
| Mean | | 45.1 Units | | 30.9 Units | |
| SD | | 8.7 Units | | 7.6 Units | |
| **'p'** | | **<0.0001 Significant** | | | |

**Figure 4 : Consumption of alcohol & hearing loss among alcoholics**

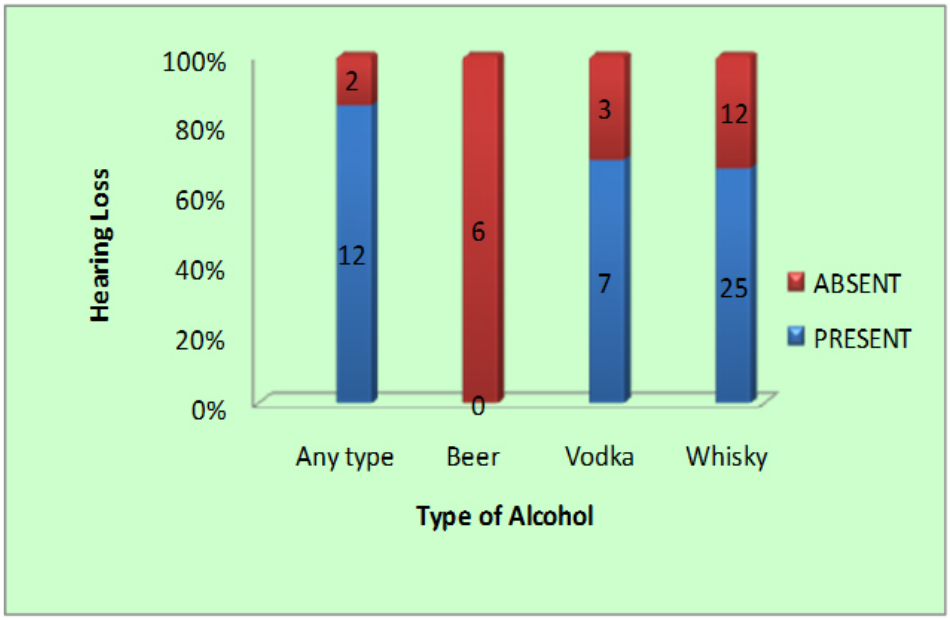



There is a significant (P<0.001) hearing loss among the alcoholics in relation to the consumption (units) of alcohol intake according to the above statistical analysis of audiometry recordings.

**Table 5 : Effect of type of alcohol on hearing**

| Type of alcohol | Number of Subjects | Hearing Loss | | | |
|---|---|---|---|---|---|
| | | Present | | Absent | |
| | | No | % | No | % |
| Any type | 14 | 12 | 85.7 | 2 | 14.3 |
| Beer | 6 | - | - | 6 | 100 |
| Vodka | 10 | 7 | 70 | 3 | 30 |
| Whisky | 37 | 25 | 67.6 | 12 | 32.4 |

**Figure 5 : Type of alcohol & hearing loss among alcoholics**

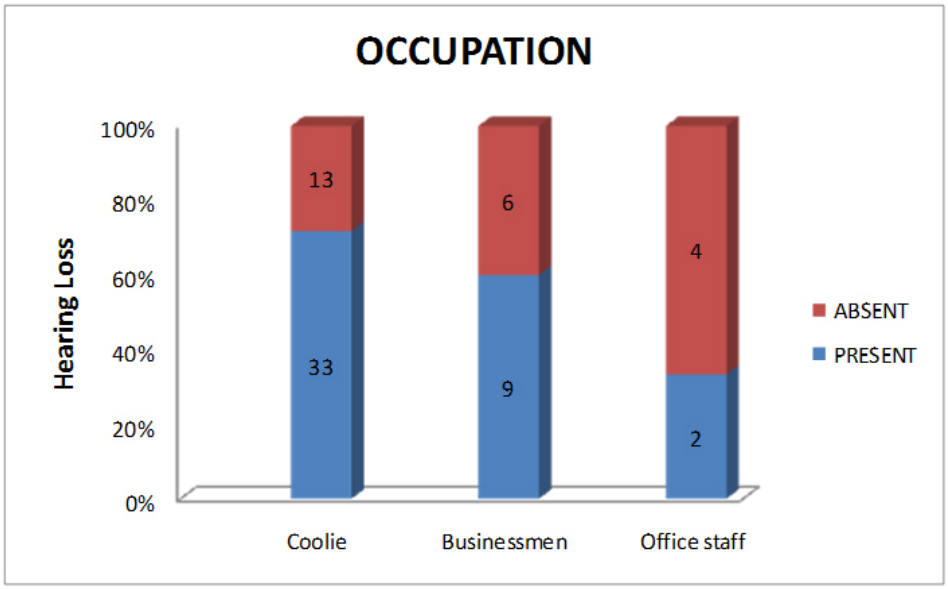



According to the study there is more sensorineural hearing loss among the alcoholics who consume any type of alcohol, which includes liquor and less with the intake of beer.

**Table 6 : Effect of occupation on hearing**

| Occupation | Number of Subjects | Hearing Loss | | | |
|---|---|---|---|---|---|
| | | Present | | Absent | |
| | | Mean | SD | Mean | SD |
| Coolie | 46 | 33 | 71.7 | 13 | 28.3 |
| Businessmen | 15 | 9 | 60 | 6 | 40 |
| Office staff | 6 | 2 | 33.3 | 4 | 66.7 |

**Figure 6 : Occupation & hearing loss among alcoholic**

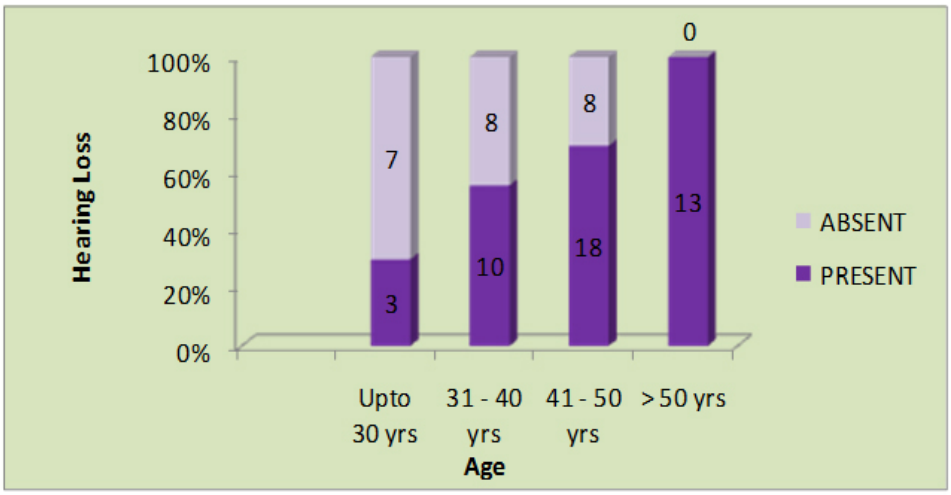



According to the above statistical analysis, alcohol intake causes more hearing loss in coolies than persons with other occupations.

**Table 7 : Effect of Age on hearing among Alcoholics**

| Group | Number of Subjects | Hearing Loss | | | |
|---|---|---|---|---|---|
| | | Present | | Absent | |
| | | No. | % | No. | % |
| Up to 30 yrs | 10 | 3 | 30 | 7 | 70 |
| 31 – 40 yrs | 18 | 10 | 55.6 | 8 | 44.4 |
| 41 – 50 yrs | 26 | 18 | 69.2 | 8 | 30.8 |
| Above 50 yrs | 13 | 13 | 100 | - | - |
| Mean | | 44.9 yrs | | 36.3 yrs | |
| SD | | 8.0 yrs | | 7.4 yrs | |
| **'p'** | | **0.0001 Significant** | | | |

**Figure 7 : Age & Hearing loss among Alcoholics**

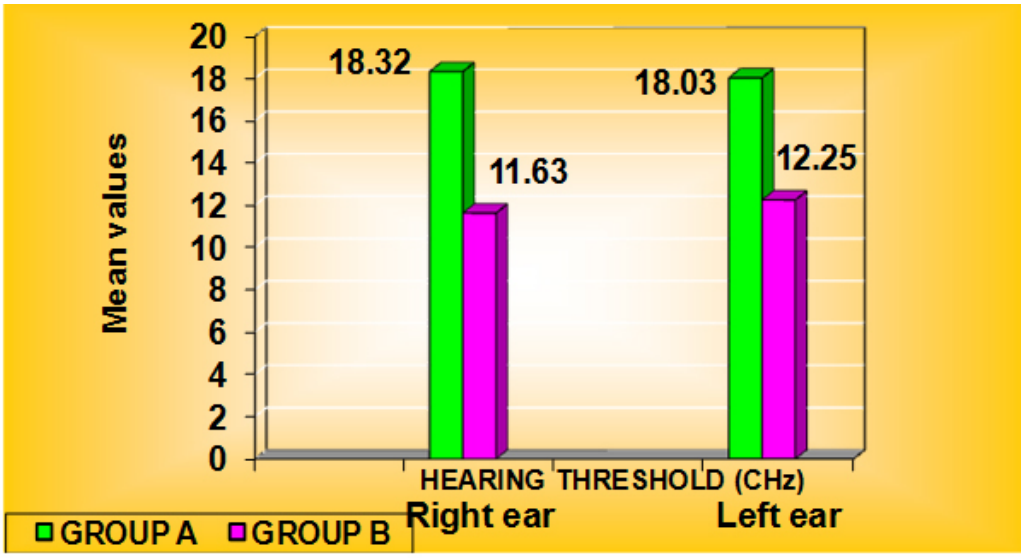



According to the association with the age, all of the alcoholics above 50 years (100%) were affected, when compared with the alcoholics who were below 30 years (30%).

**Table 8 : Hearing Threshold in Alcoholics and Non-Alcoholics**

| Group | Hearing Threshold (Hz) | | | |
|---|---|---|---|---|
| | Right ear | | Left ear | |
| | Mean | SD | Mean | SD |
| Group A (Alcoholics) | 18.32 | 5.73 | 18.03 | 5.5 |
| Group B (Non-Alcoholics) | 11.63 | 2.54 | 12.25 | 2.39 |
| **'p'** | **<0.0001 Significant** | | **<0.0001 significant** | |

**Figure 8 : Hearing threshold (Hz)**

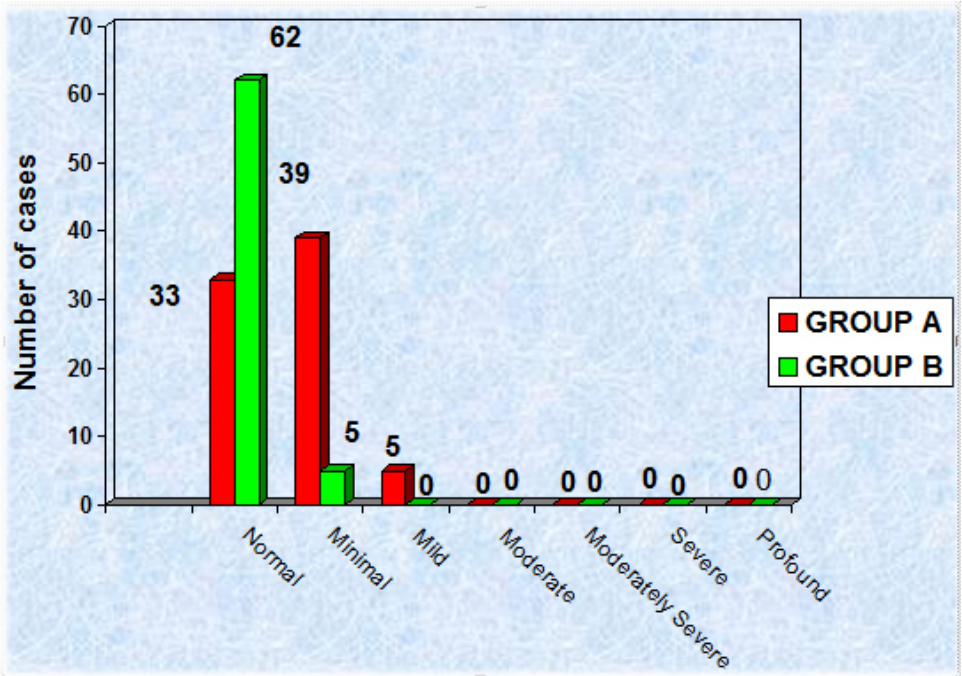



The hearing threshold of the alcoholics had increased and causes bilaterally equal minimal to mild hearing loss. The right ear was slightly more affected in the alcoholics when compared with the left ear.

**Table 9 : Hearing loss among Alcoholics and Non-Alcoholics**

| Hearing Loss | Group A | Group B |
|---|---|---|
| Normal hearing | 33 | 62 |
| Minimal hearing loss | 39 | 5 |
| Mild hearing loss | 5 | 0 |
| Moderate hearing loss | 0 | 0 |
| Moderately severe loss | 0 | 0 |
| Severe hearing loss | 0 | 0 |
| Profound deafness | 0 | 0 |

**Figure 9 : Degree of Hearing loss**




The above table shows the intake of alcohol causes minimal to mild hearing loss when compared to the non-alcoholics.

# *DISCUSSION*

# DISCUSSION

The present study of the impact of alcohol on auditory thresholds involves 67 alcoholic males compared with 67 age matched non-alcoholic controls in the age group of 25 to 55 years and who had consumed alcohol for more than 2 years. The study was done to analyze the alcoholic effects, with the help of pure tone audiometer and DPOAE test.

This study shows that there is a significant association ('p' value <0.0001) with alcohol intake with hearing impairment. According to the pure tone audiometry findings, 65.7% of alcoholics were with hearing impairment, when compared with the controls who had only 7.5% affected. By using DPOAE it was found that 62.7% of alcoholics were affected by hearing loss, while only 7.5% of non-alcoholics were affected. The higher frequencies were more affected when compared with the lower frequencies.

Sandra Beatriz et al[9], Kavitha Ashok Kumar et al[52], Tahwinder Upile et al[7], Marcieli Belle et al[70], Perez et al[50] have observed the auditory effects of alcohol and suggested that there is a strong relationship between alcohol and hearing loss.

Kavitha Ashok Kumar et al found that absence of emissions in 76.6% of alcoholics in their DPOAE test[52], suggesting that alcohol causes damage to the outer hair cells of the cochlea and also proved that the sensorineural hearing loss was seen in high frequencies like 4000Hz to 8000Hz. Due to the high frequency loss which was above the normal speech frequency, the alcoholics never complained of hearing loss, because they didn't realize the hearing impairment.

J.H.Hwang et al demonstrated that *DPOAE amplitudes in higher frequencies were decreased* after moderate alcohol consumption (1.2 g/Kg) [51]. The audiological assessment done by Sandra Beatriz et al[11] using puretone audiometry proved that higher frequencies above 3000Hz were more affected in alcoholics and also found the absence of TEOAE recordings more in alcoholic subjects than compared to the controls. The study by Roshan K Varma also confirms the elevation of auditory thresholds in the alcoholic subjects[80]. In contrast Tahwinder Upile et al suggested that the lower frequencies like 1000Hz were also affected by alcohol which was the frequency to discriminate vowels[7].

The present study indicates that the ***duration of consumption of alcohol also influences the hearing loss***. Whenever the duration of intake of alcohol increases, hearing impairment also increases ('p' value <0.0001 Significant). Among the study group, 22.2% of cases had hearing loss in case of less than 5 years duration, but almost all the 7 cases (100%) were affected who had more than 20 years of alcohol intake. These findings are similar to that of Sandra Beatriz et al[11], Marcieli Belle et al[70], Golabek et al[76,] and Wheeler et al[78] who observed more incidences of hearing impairment and high frequency hearing loss in subjects who consumed alcohol for longer periods[11, 70]. In contrast, Kavitha Ashok Kumar et al[52] and Rossi et al[84] found that there was no relationship between duration of alcohol and severity of hearing loss. The hearing loss reported may be due to the long term exposure to alcohol resulting in an ototoxicity affecting cochlear function harming specially the external hair cells.

The study showed that there is a greater significant relationship ('p' value <0.0001) between the ***amount of consumption of alcohol and hearing loss***.

When intake of alcohol was less than 30 units, only 5.6% of alcoholics got hearing loss and the severity is increases proportionate with the increase of intake of alcohol. Almost all(9) of the cases (100%) had hearing loss when they consumed more than 50 units/week. According to the classification of Royal college of physicians, psychiatrists and general practitioners, low risk for men who consume alcohol up to 21units per week, increased risk for men consuming 22 units to 50 units per week and high risk for men who consume above 50 units/week[3].

Juen-Haur Hwang et al suggested that the DPOAE changes produced by alcohol were concentration dependent[51]. Tahwinder Upile et al showed that moderate consumption of alcohol affected the hearing thresholds by producing dullness to the puretones in speech frequencies[7]. In contrast, Kavitha Ashok Kumar et al found that there was no relationship between quantity of consumption of alcohol and severity of hearing loss[52].

The ***type of alcohol also influences the hearing loss***. The alcoholics who consumed only beer were not affected by hearing loss.

The reason might be the alcohol content of beer was less when compared with the other types. Those who consumed various forms of alcohol [which include beer or whisky or vodka or rum or gin or liquor (spirits) or any other alcoholic beverage whatever he got, at that time] were more affected. This finding was correlated with the prospective study of alcohol use and hearing loss in men by Sharon G. Curgan et al, in which they suggested that there was increased risk of hearing impairment in those who consumed liquor (spirits)[9]. The effect might be due to the increased concentration of alcohol content in liquor.

There is some relationship between the ***hearing loss and occupation of alcoholics.*** The coolies, were more affected by hearing loss. This might be associated with the fact that due to financial problems they took only alcohol and without any food, they went for sleep. Sharon G. curgan et al suggested that the alcoholics, those who consume higher amounts of alcohol with lower intake of Vitamin $B_{12}$ and folic acid were associated with higher risk of hearing impairment[9]. Lohle E et al found that vitamin A deficiency in the chronic alcoholics causes poor hearing[10].

In relation with ***age and alcoholic hearing loss***, only few alcoholics with earlier ages were affected, whereas alcohol affected most of the older aged persons. According to the Marcieli Belle et al study, they proved older individuals (alcoholics) suffered hearing loss more, when compared to younger ones[70].

Alcohol not only affects the central nervous system, but also affected the functions of outer hair cells. The possibilities of outer hair cell damage caused in alcoholism, such as

1. Alcohol and its metabolites causes disturbances in the endocochlear environment and abnormal outer hair cell motility[51].

2. Alcohol enhances the inhibitory transmission via $GABA_A$ type receptors and suppresses the excitatory transmission via N-Methyl D-aspartate receptors[4, 51].

3. Alcohol affects the middle ear muscles and thereby affects the acoustic reflex thresholds[51, 52].

Prestin is a trans membrane protein that mechanically contracts and elongates leading to electro motility of outer hair cells (OHC).

Electro motility is the driving force behind the somatic motor of the cochlear amplifier, which is a mammalian evolution that increases sensitivity to incoming sound wave frequencies, and thus amplifies the signal. Prestin acts through an intrinsic voltage-sensor (IVS) in which intracellular chloride binds allosterically to prestin to modify the shape.

Previous experiments have indicated that prestin sensitive to the levels of cholesterol in the cell membrane. Also known modulator of lipid bi-layer could have the potential to have the impact on prestin function. Prestin is necessary for the cochlear amplification and electro motility of the outer hair cell[37]. Alcohol modulated the lipid bilayer properties and could have an impact on prestin function[56].

Toxic effects of alcohol occurred in the basal turns of the cochlea[11]. The absence of otoacoustic emissions, which indicated that the damage occurred in outer hair cells in the organ of corti. Alcohol acts centrally by involving the temporal and binaural summation of auditory signals and also peripherally by acting on outer hair cells[7].

The acute changes in hearing loss were completely reversible, whereas chronic alcoholic changes were irreversible[7,51]. Long-term exposure to alcohol abuse causes ototoxicity, which affect cochlear function, particularly by damaging the external hair cells[10].

DPOAE measurements were more sensitive than conventional audiometry for detecting the minor and early changes in the outer hair cells[51].

This study confirms that consumption of alcohol leads to sensorineural hearing loss for higher frequency sounds.

# *SUMMARY*

## SUMMARY

- This study shows alcoholic individuals have hearing loss.
- The hearing loss caused by long term consumption of alcohol is minimal to mild sensorineural hearing loss.
- Alcohol abusers have a high frequency hearing loss.
- There was a significant difference in sensorineural hearing loss between alcoholics and Non-alcoholics.
- The prevalence of sensorineural hearing loss among alcoholics was observed to be 65%.
- A positive correlation was documented between duration of consumption of alcohol and sensorineural hearing loss.
- There was a proportionate increase in the hearing impairment and quantity of the intake of alcohol.
- There was a significant difference between the type of alcoholic beverages and the hearing loss.
- Association of alcohol and age of the alcoholics also had the significant relationship between them.
- There was only a slight difference between the hearing thresholds of right and left ears. The sensorineural hearing loss caused by alcohol was bilaterally symmetrical.

# *CONCLUSION*

# CONCLUSION

Alcohol is one of the leading causes of death in human beings. It affects all systems of the body, including the special senses like hearing. Alcohol affects hearing not only via the central nervous system, but also influences the function of outer hair cells. The study also suggested that alcohol affects the central auditory system by inhibiting the excitatory transmission via N-Methyl D-aspartate receptors. Alcohol affects the peripheral auditory system by altering the prestin, a motor protein in the outer hair cells and reduces the cochlear amplification and electro motility of the outer hair cell.

Hearing loss can interfere with the quality of life, restricting the ability to interact with others. This leads to stress and misunderstandings in communications. Hearing impairment filtering out sound experiences, which gives pleasure and meaning to life. The study confirms the positive correlation between alcoholism and hearing loss.

“Prevention is better than cure”. Early detection of hearing impairment among the alcoholics is essential as appropriate management can improve their quality of life.

The outcome of this study will raise the awareness among the alcoholics regarding the hearing loss and prevent the further progression of hearing impairment.

For prevention of hearing loss in alcoholics, the first step is the abstinence of alcohol by giving health education regarding the risks by outpatient counseling[85]. This study suggests that PTA and DPOAE tests are the basic and gold standard tests to detect the hearing loss in early stage, so as to start the treatment earlier and cure the damage to the auditory pathway. In later periods the hearing loss will be incurable.

**LIMITATION:**

In the present study sensorineural hearing impairment is detected by basic screening by using pure tone audiometry and DPOAEs. The early signs of hearing loss could be detected by high frequency audiometry which is more sensitive and accurate than conventional audiometry. Pure tone and speech audiometry or Brainstem evoked response audiometry may help to further demonstrate the actual hearing pathways (central and/or peripheral) affected by alcohol.

A longitudinal study with a large sample size involving the women, younger men, and other racial groups will be of great value to demonstrate the relation between alcohol intake and hearing impairment.

**FUTURE SCOPE OF PRESENT STUDY:**

Additional studies are needed to demonstrate the role of vitamin A and vitamin B12 in alcohol-related ototoxicity. A study with a greater numbers of subjects with measurement of both breath and blood alcohol levels would lead to more accuracy and scientific validation of results.

Despite more progress, there is a lot left to understand the structural and molecular mechanism underlying the effect of alcohol on auditory pathways. In the treatment aspect, in the near future, cochlear gene therapy may be used in the treatment of sensorineural hearing loss. Regeneration of hair cells using viral vectors may become the best treatment method of hearing loss.

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# *ANNEXURES*

**AUDIT ; The Alcohol Use Disorders Identification Test**

Total scores of 8 or more are indicators of harmful alcohol use, and also alcohol dependence possibility.

**Interpretation**

Scores between 8 and 15 are advice to reduce drinking.

Scores between 16 and 19 need counseling and monitoring.

Scores of 20 or above clearly indicate diagnostic evaluation for alcohol dependence.

**Standard drink (S drink)**

1 s drink in the UK: 8 g

1 s drink in the USA: 14 g

**Beverages**

Beer contained between 2% and 5% volume by volume of pure alcohol. Wines contained 10.5% to 18.9%, Spirits varied from 24.3% to 90% of pure ethanol. For example,

1 can beer (330 ml) at 5% x (strength) 0.79 (conversion factor) = 13 grams of ethanol.

1 glass wine (140 ml) at 12% x 0.79 = 13.3 grams of ethanol

1 shot spirits (40 ml) at 40% x 0.79 = 12.6 grams of ethanol.

Details about typical amounts of alcohol contained in various beverages

| Drink | Typical ABV |
|---|---|
| Fruit juice (naturally occurring) | less than 0.1% |
| beer | 2%–12% (usually 4%–6%) |
| Wine | 9%–16% (most often 12.5%–14.5%)[7] |
| Vodka | 35%–50% (usually 40%, minimum of 37.5% in the European Union) |
| Brandy | 35%–60% (usually 40%) |
| Rum | 37.5%–80% |
| Gin | 40%–50% |
| Whisky | 40%–68% (usually 40%, 43% or 46%) |
| Spirit | 95%-96% |

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| Whisky | 40%–68% (usually 40%, 43% or 46%) |
| Spirit | 95%-96% |

# CONSENT FORM

Dr. N. Sivakumar, Post Graduate student in the Department of Physiology, Coimbatore Medical College, Coimbatore, is studying the "Impact of Alcohol on Auditory threshold". Procedure was explained to me clearly.

I hereby give my consent to participate in this study. The data obtained herein may be used for research and publication.

Name :

Place :

Signature :

# PROFORMA

Serial No : Date:

Name :

Age :

Sex :

Occupation :

Address : Phone no.

Presenting illness

H/O Vertigo :

H/O Tinnitus :

H/O Hard of hearing : Yes / No

Personal history

H/O Alcohol intake : Yes / No

If yes, Duration :

Type :

How often :

Units of alcohol/week :

H/O Cigarette smoking : Yes / No

H/O Drug abuse : Yes / No

H/O Exposure to noisy surroundings : Yes / No

Past history

H/O Systemic hypertension : Yes / No

H/O Diabetes mellitus : Yes / No

H/O Ear diseases : Yes / No

H/O Nervous system disorders : Yes / No

H/O Intake of any ototoxic drugs : Yes / No

Family history

H/O Systemic hypertension : Yes / No

H/O Diabetes mellitus : Yes / No

H/O Hereditary disorders in family : Yes / No

**ON EXAMINATION**

Built :

Height :

Weight :

Pulse rate :

Blood pressure :

**GENERAL EXAMINATION**

Pallor :

Icterus :

Clubbing :

Cyanosis :

Lymphadenopathy :

Pedal edema :

## SYSTEMIC EXAMINATION

Cardio vascular system :

Respiratory system :

Central nervous system :

## EXAMINATION OF EAR

| **Otoscopy** | | |
|---|---|---|
| | **RIGHT** | **LEFT** |
| Presence of wax<br>Ear discharge<br>Any other gross anomaly<br>Rinne's test<br>Weber's test | | |
| **Investigation**<br>Pure tone audiometry<br>DPOAE | | |

# *MASTER CHART*

## Controls

| S.No | Name | Age | Occupation | Alcoholic | RTR | RTL | Webers Test | HTR | HTL | PTA | PTAR | PTAL | DPOAE |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | Selvaraj | 35 | Cooly | No | positive | positive | Centralised | 10 | 13.3 | No | no | No | Pass |
| 2 | Gothandam | 46 | Cooly | No | positive | positive | Centralised | 13.3 | 13.3 | No | no | No | Pass |
| 3 | Suresh | 54 | Cooly | No | positive | positive | Centralised | 14.3 | 13.3 | No | no | No | Pass |
| 4 | Muthu kumar | 39 | Cooly | No | positive | positive | Centralised | 13.3 | 14.6 | No | no | No | Pass |
| 5 | Vijayakumar | 44 | Office | No | positive | positive | Centralised | 10.6 | 13.3 | No | no | No | Pass |
| 6 | Govinda samy naidu | 55 | Cooly | No | positive | positive | Centralised | 18.3 | 18.3 | Yes | yes | Yes | Refer |
| 7 | Daniel | 34 | Cooly | No | positive | positive | Centralised | 8.3 | 13.3 | No | no | No | Pass |
| 8 | Gopala krishnan | 41 | Cooly | No | positive | positive | Centralised | 10 | 11.6 | No | no | No | Pass |
| 9 | Nallasamy | 53 | Cooly | No | positive | positive | Centralised | 11.3 | 12.6 | No | no | No | Pass |
| 10 | Dharmaraj | 50 | Business | No | positive | positive | Centralised | 12.6 | 13.3 | No | no | No | Pass |
| 11 | Vikraman | 45 | Office | No | positive | positive | Centralised | 12 | 12 | No | no | No | Pass |
| 12 | Thevana Goundar | 50 | Cooly | No | positive | positive | Centralised | 13.3 | 13.3 | No | no | No | Pass |
| 13 | Chenniappan | 50 | Cooly | No | positive | positive | Centralised | 13.3 | 13.3 | No | no | No | Pass |
| 14 | Vincent paul | 49 | Cooly | No | positive | positive | Centralised | 14 | 13.3 | No | no | No | Pass |
| 15 | Sivakumar | 35 | Cooly | No | positive | positive | Centralised | 8.3 | 8.3 | No | no | No | Pass |
| 16 | Vikesh | 53 | Cooly | No | positive | positive | Centralised | 13.3 | 12.6 | No | no | No | Pass |
| 17 | Viswanathan | 52 | Cooly | No | positive | positive | Centralised | 8.3 | 11.6 | No | no | No | Pass |
| 18 | muthu paramasivam | 46 | Cooly | No | positive | positive | Centralised | 11 | 13.3 | No | no | No | Pass |
| 19 | Jeyaprakash | 43 | Business | No | positive | positive | Centralised | 13.3 | 13.3 | No | no | No | Pass |
| 20 | Venkatachalam | 41 | Business | No | positive | positive | Centralised | 10 | 12.6 | No | no | No | Pass |
| 21 | Rajesh | 33 | Cooly | No | positive | positive | Centralised | 11.6 | 10 | No | no | No | Pass |
| 22 | Prem | 36 | Cooly | No | positive | positive | Centralised | 8.3 | 13.3 | No | no | No | Pass |
| 23 | Ayyannasamy | 55 | Cooly | No | positive | positive | Centralised | 13.3 | 15 | No | no | No | Pass |
| 24 | Saravanakumar | 36 | Cooly | No | positive | positive | Centralised | 10 | 13.3 | No | no | No | Pass |
| 25 | Natraj | 32 | Cooly | No | positive | positive | Centralised | 11.6 | 13.3 | No | no | No | Pass |

PTAR - Pure Tone Audiometry Notch Right Ear
RTR - Rinne's Test Right Ear, RTL - Rinne's Test Left Ear
PTAL - Pure Tone Audiometry Notch Left Ear
PTA - Pure Tone Audiometry Notch,
HTR - Hearing Threshold Right Ear, HTL Hearing Threshold Left Ear

| | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 26 | Govindaraj | 39 | Office | No | positive | positive | Centralised | 8.3 | 11.6 | No | no | No | Pass |

| | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 27 | Lakshmanan | 44 | Business | No | positive | positive | Centralised | 13.3 | 13.3 | No | no | No | Pass |
| 28 | Abdul jaffer | 54 | Office | No | positive | positive | Centralised | 13.3 | 12.6 | No | no | No | Pass |
| 29 | Stanley | 51 | Cooly | No | positive | positive | Centralised | 11.6 | 11.6 | No | no | No | Pass |
| 30 | Anandaraj | 41 | Cooly | No | positive | positive | Centralised | 10 | 15 | No | no | No | Pass |
| 31 | John | 52 | Cooly | No | positive | positive | Centralised | 13.3 | 8.3 | No | no | No | Pass |
| 32 | Thangaraj | 47 | Cooly | No | positive | positive | Centralised | 13.3 | 15 | No | no | No | Pass |
| 33 | Rajavel murugan | 48 | Cooly | No | positive | positive | Centralised | 12 | 13.3 | No | no | No | Pass |
| 34 | Abdhul | 30 | Cooly | No | positive | positive | Centralised | 11.6 | 8.3 | No | no | No | Pass |
| 35 | Ramesh | 45 | Cooly | No | positive | positive | Centralised | 13.3 | 13.3 | No | no | No | Pass |
| 36 | Rajakumar | 37 | Cooly | No | positive | positive | Centralised | 13.3 | 14 | No | no | No | Pass |
| 37 | Praveen | 31 | Cooly | No | positive | positive | Centralised | 11.6 | 13.3 | No | no | No | Pass |
| 38 | Kannan | 51 | Cooly | No | positive | positive | Centralised | 15 | 13.3 | No | no | No | Pass |
| 39 | Srikanth | 26 | Cooly | No | positive | positive | Centralised | 8.3 | 11.6 | No | no | No | Pass |
| 40 | Vijay | 28 | Cooly | No | positive | positive | Centralised | 8.3 | 13.3 | No | no | No | Pass |
| 41 | Appasamy | 45 | Office | No | positive | positive | Centralised | 9 | 8.3 | No | no | No | Pass |
| 42 | Pazhani | 49 | Business | No | positive | positive | Centralised | 8.3 | 8.3 | No | no | No | Pass |
| 43 | Appachi | 53 | Cooly | No | positive | positive | Centralised | 18.3 | 18.3 | Yes | Yes | Yes | Refer |
| 44 | Saravanan | 37 | Cooly | No | positive | positive | Centralised | 13.3 | 13.3 | No | no | No | Pass |
| 45 | Ambethkumar | 29 | Cooly | No | positive | positive | Centralised | 8.3 | 13.3 | No | no | No | Pass |
| 46 | Ashok raja | 32 | Cooly | No | positive | positive | Centralised | 11.6 | 11.6 | No | no | No | Pass |
| 47 | Kumaravel | 53 | Business | No | positive | positive | Centralised | 16.3 | 13.3 | No | no | No | Pass |
| 48 | Eswara raja | 52 | Business | No | positive | positive | Centralised | 10 | 8.3 | No | no | No | Pass |
| 49 | Thenappan | 38 | Cooly | No | positive | positive | Centralised | 8.3 | 13.3 | No | no | No | Pass |
| 50 | Sivaraja | 40 | Cooly | No | positive | positive | Centralised | 11.6 | 8.3 | No | no | No | Pass |
| 51 | Ibrahim | 47 | Office | No | positive | positive | Centralised | 13.3 | 13.3 | No | no | No | Pass |
| 52 | Jeya singh | 50 | Cooly | No | positive | positive | Centralised | 12 | 12 | No | no | No | Pass |
| 53 | Jothi kannan | 25 | Cooly | No | positive | positive | Centralised | 8.3 | 8.3 | No | no | No | Pass |

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| | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 54 | Senthil murugan | 27 | Cooly | No | positive | positive | Centralised | 8.3 | 11.6 | No | no | No | Pass |

| | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 55 | Mubarak ali | 36 | Cooly | No | positive | positive | Centralised | 11.6 | 11.6 | No | no | No | Pass |
| 56 | Reji ebenezar | 43 | Cooly | No | positive | positive | Centralised | 8.3 | 8.3 | No | no | No | Pass |
| 57 | Muthuraj | 40 | Cooly | No | positive | positive | Centralised | 14 | 13.3 | No | no | No | Pass |
| 58 | Shanmugha velu | 30 | Cooly | No | positive | positive | Centralised | 13.3 | 13.3 | No | no | No | Pass |
| 59 | Shadik Batcha | 52 | Cooly | No | positive | positive | Centralised | 15 | 13.3 | No | no | No | Pass |
| 60 | Pazhanisamy | 43 | Cooly | No | positive | positive | Centralised | 13.3 | 8.3 | No | no | No | Pass |
| 61 | Mohan raj | 34 | Cooly | No | positive | positive | Centralised | 10 | 8.3 | No | no | No | Pass |
| 62 | Narayanan | 38 | Cooly | No | positive | positive | Centralised | 13.3 | 13.3 | No | no | No | Pass |
| 63 | Rajkumar | 46 | Cooly | No | positive | positive | Centralised | 8.3 | 8.3 | No | no | No | Pass |
| 64 | Abdul ali | 37 | Cooly | No | positive | positive | Centralised | 8.3 | 8.3 | No | no | No | Pass |
| 65 | Karthikeyan | 42 | Cooly | No | positive | positive | Centralised | 13.3 | 13.3 | No | no | No | Pass |
| 66 | Paneer selvam | 38 | Cooly | No | positive | positive | Centralised | 8.3 | 13.3 | No | no | No | Pass |
| 67 | Gopikrishna | 48 | Cooly | No | positive | positive | Centralised | 13.3 | 13.3 | No | no | No | Pass |

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PTAL - Pure Tone Audiometry Notch Left Ear
PTA - Pure Tone Audiometry Notch,
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## Cases

| S.No | Name | Age | Occupation | Alcoholic | Duration | Type | Unit | RTR | RTL | Webers Test | HTR | HTL | PTA notch | PTAR | PTAL | DPOAE |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | Subramanian | 32 | Cooly | yes | 3 | Any | 30 | positive | positive | Centralised | 13 | 13 | Absent | Absent | Absent | Pass |
| 2 | Muniappan | 55 | Cooly | yes | 25 | Whisky | 40 | positive | positive | Centralised | 18 | 18 | Present | Present | Present | Refer |
| 3 | Shanmugham | 50 | Cooly | yes | 20 | Whisky | 60 | positive | positive | Centralised | 23 | 20 | Present | Present | Present | Refer |
| 4 | Shanmugha velu | 49 | Business | yes | 19 | Vodga | 40 | positive | positive | Centralised | 18 | 23 | Present | Present | Present | Refer |
| 5 | Murugan | 44 | Cooly | yes | 12 | Whisky | 35 | positive | positive | Centralised | 13 | 18 | Present | Absent | Present | Pass |
| 6 | Aniruthan | 40 | Cooly | yes | 11 | Whisky | 50 | positive | positive | Centralised | 20 | 13 | Present | Present | Absent | Refer |
| 7 | Kannappan | 34 | Cooly | yes | 6 | Any | 45 | positive | positive | Centralised | 18 | 15 | Present | Present | Absent | Refer |
| 8 | Nehru | 43 | Cooly | yes | 12 | Whisky | 40 | positive | positive | Centralised | 18 | 18 | Present | Present | Present | Refer |
| 9 | Krishnakumar | 51 | Business | yes | 21 | Vodga | 50 | positive | positive | Centralised | 33 | 28 | Present | Present | Present | Refer |
| 10 | Anandhan | 48 | Cooly | yes | 20 | Whisky | 60 | positive | positive | Centralised | 22 | 20 | Present | Present | Present | Refer |
| 11 | Balamurugan | 39 | Cooly | yes | 11 | Any | 55 | positive | positive | Centralised | 18 | 18 | Present | Present | Present | Refer |
| 12 | Narendran | 30 | Cooly | yes | 7 | Whisky | 25 | positive | positive | Centralised | 8.3 | 8.3 | Absent | Absent | Absent | Pass |
| 13 | Krishnan kutti | 50 | Business | yes | 18 | Whisky | 35 | positive | positive | Centralised | 18 | 18 | Present | Present | Present | Refer |
| 14 | Senthil kumar | 55 | Business | yes | 22 | Whisky | 60 | positive | positive | Centralised | 18 | 18 | Present | Present | Present | Refer |
| 15 | Koni | 37 | Office | yes | 10 | Beer | 25 | positive | positive | Centralised | 12 | 12 | Absent | Absent | Absent | Pass |
| 16 | Arun | 46 | Business | yes | 13 | Whisky | 40 | positive | positive | Centralised | 13 | 13 | Absent | Absent | Absent | Pass |
| 17 | Senthil kannan | 48 | Cooly | yes | 20 | Any | 45 | positive | positive | Centralised | 23 | 28 | Present | Present | Present | Refer |
| 18 | Iyyappan | 35 | Cooly | yes | 8 | Any | 40 | positive | positive | Centralised | 20 | 15 | Present | Present | Absent | Pass |
| 19 | Nagaraj | 42 | Cooly | yes | 11 | Beer | 25 | positive | positive | Centralised | 13 | 13 | Absent | Absent | Absent | Pass |
| 20 | Siva | 43 | Business | yes | 15 | Vodga | 60 | positive | positive | Centralised | 18 | 20 | Present | Present | Present | Refer |
| 21 | Nehru | 28 | Cooly | yes | 4 | Whisky | 30 | positive | positive | Centralised | 8.3 | 10 | Absent | Absent | Absent | Pass |
| 22 | Kumar | 45 | Office | yes | 15 | Vodga | 25 | positive | positive | Centralised | 13 | 12 | Absent | Absent | Absent | Pass |
| 23 | Vasu | 36 | Cooly | yes | 9 | Any | 50 | positive | positive | Centralised | 18 | 20 | Present | absent | Present | Refer |
| 24 | Vijaya ragavan | 50 | Cooly | yes | 15 | Whisky | 35 | positive | positive | Centralised | 18 | 18 | Present | Present | Present | Refer |
| 25 | Navas | 55 | Cooly | yes | 27 | Any | 60 | positive | positive | Centralised | 30 | 28 | Present | Present | Present | Refer |
| 26 | Gothandaraman | 27 | Business | yes | 3 | Whisky | 25 | positive | positive | Centralised | 15 | 13 | Absent | Absent | Absent | Pass |
| 27 | Samaran | 31 | Business | yes | 8 | Beer | 25 | positive | positive | Centralised | 8.3 | 10 | Absent | Absent | Absent | Pass |
| 28 | Anvar | 50 | Cooly | yes | 20 | Any | 60 | positive | positive | Centralised | 25 | 28 | Present | Present | Present | Refer |
| 29 | Ramar | 42 | Office | yes | 12 | Whisky | 30 | positive | positive | Centralised | 15 | 15 | Absent | Absent | Absent | Pass |

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PTAL - Pure Tone Audiometry Notch Left Ear
PTA - Pure Tone Audiometry Notch,
HTR - Hearing Threshold Right Ear, HTL Hearing Threshold Left Ear
Unit -Unit of Alcohol per week

| | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 30 | Jegannathan | 47 | Office | yes | 17 | Vodga | 25 | positive | positive | Centralised | 13 | 13 | Absent | Absent | Absent | Pass |
| 31 | Mari kannu | 43 | Cooly | yes | 12 | Whisky | 40 | positive | positive | Centralised | 18 | 15 | Present | Present | Absent | Refer |
| 32 | Thanush kodi | 44 | Cooly | yes | 16 | Any | 40 | positive | positive | Centralised | 23 | 20 | Present | Present | Present | Refer |
| 33 | Ramakrishnan | 27 | Cooly | yes | 5 | Whisky | 35 | positive | positive | Centralised | 15 | 10 | Absent | Absent | Absent | Pass |
| 34 | Anbu thambi | 50 | Cooly | yes | 20 | Whisky | 45 | positive | positive | Centralised | 20 | 18 | Present | Present | Present | Refer |
| 35 | Guru batham | 47 | Cooly | yes | 18 | Whisky | 40 | positive | positive | Centralised | 23 | 23 | Present | Present | Present | Refer |
| 36 | Muthamizhan | 29 | Cooly | yes | 5 | Whisky | 50 | positive | positive | Centralised | 20 | 20 | Present | Present | Present | Refer |
| 37 | Stanley | 45 | Cooly | yes | 13 | Any | 25 | positive | positive | Centralised | 18 | 18 | Present | Present | Present | Refer |
| 38 | Ragu nanthanam | 41 | Business | yes | 12 | Beer | 30 | positive | positive | Centralised | 13 | 13 | Absent | Absent | Absent | Pass |
| 39 | Parameshwaran | 36 | Cooly | yes | 8 | Whisky | 30 | positive | positive | Centralised | 13 | 13 | Absent | Absent | Absent | Pass |
| 40 | Senthil kumar | 29 | Cooly | yes | 5 | Whisky | 40 | positive | positive | Centralised | 8.3 | 13 | Absent | Absent | Absent | Pass |
| 41 | Karthikeyan | 39 | Cooly | yes | 7 | Any | 45 | positive | positive | Centralised | 20 | 20 | Present | Present | Present | Refer |
| 42 | Pavithran | 39 | Cooly | yes | 9 | Beer | 25 | positive | positive | Centralised | 12 | 13 | Absent | Absent | Absent | Pass |
| 43 | Hareesh | 46 | Office | yes | 16 | Vodga | 35 | positive | positive | Centralised | 18 | 18 | Present | Present | Present | Refer |
| 44 | George | 54 | Cooly | yes | 19 | Whisky | 55 | positive | positive | Centralised | 18 | 20 | Present | Present | Present | Refer |
| 45 | Rangarajan | 49 | Cooly | yes | 13 | Whisky | 35 | positive | positive | Centralised | 15 | 13 | Absent | Absent | Absent | Pass |
| 46 | Kathirvel | 55 | Cooly | yes | 20 | Whisky | 55 | positive | positive | Centralised | 23 | 23 | Present | Present | Present | Refer |
| 47 | Dhandabani | 46 | Office | yes | 14 | Vodga | 35 | positive | positive | Centralised | 18 | 18 | Present | Present | Present | Refer |
| 48 | Maria joseph | 26 | Business | yes | 5 | Whisky | 30 | positive | positive | Centralised | 8.3 | 10 | Absent | Absent | Absent | Pass |
| 49 | Krishna kumar | 34 | Cooly | yes | 8 | Beer | 25 | positive | positive | Centralised | 13 | 13 | Absent | Absent | Absent | Pass |
| 50 | Balusamy | 37 | Cooly | yes | 10 | Whisky | 45 | positive | positive | Centralised | 20 | 18 | Present | Present | Present | Refer |
| 51 | Saravanan | 42 | Business | yes | 15 | Whisky | 25 | positive | positive | Centralised | 13 | 13 | Absent | Absent | Absent | Pass |
| 52 | Chandra sekhar | 40 | Cooly | yes | 12 | Any | 30 | positive | positive | Centralised | 12 | 10 | Absent | Absent | Absent | Pass |
| 53 | Gopi kannan | 52 | Cooly | yes | 18 | Whisky | 45 | positive | positive | Centralised | 23 | 20 | Present | Present | Present | Refer |
| 54 | Velusamy | 54 | Cooly | yes | 20 | Whisky | 50 | positive | positive | Centralised | 23 | 23 | Present | Present | Present | Refer |
| 55 | Jai ganesh | 27 | Cooly | yes | 16 | Whisky | 35 | positive | positive | Centralised | 20 | 18 | Present | Present | Present | Refer |
| 56 | Hydher ali | 38 | Business | yes | 10 | Any | 45 | positive | positive | Centralised | 20 | 22 | Present | Present | Present | Refer |
| 57 | Muthu | 51 | Cooly | yes | 16 | Whisky | 40 | positive | positive | Centralised | 25 | 28 | Present | Present | Present | Refer |
| 58 | Purushothaman | 53 | Business | yes | 17 | Vodga | 40 | positive | positive | Centralised | 18 | 20 | Present | Present | Present | Refer |
| 59 | Krishna | 39 | Cooly | yes | 11 | Vodga | 50 | positive | positive | Centralised | 23 | 28 | Absent | Absent | Absent | Pass |
| 60 | Vinoth | 32 | Cooly | yes | 15 | Whisky | 50 | positive | positive | Centralised | 33 | 27 | Present | Present | Present | Refer |
| 61 | Balamurugan | 37 | Cooly | yes | 14 | Whisky | 45 | positive | positive | Centralised | 18 | 20 | Present | Present | Present | Refer |
| 62 | Varatharajan | 41 | Business | yes | 15 | Vodga | 45 | positive | positive | Centralised | 20 | 18 | Present | Present | Present | Refer |
| 63 | Baskaran | 26 | Business | yes | 4 | Any | 45 | positive | positive | Centralised | 27 | 20 | Present | Present | Present | Refer |
| 64 | Pirabakaran | 52 | Cooly | yes | 25 | Whisky | 35 | positive | positive | Centralised | 20 | 22 | Present | Present | Present | Refer |
| 65 | Venkatesh | 25 | Cooly | yes | 3 | Whisky | 50 | positive | positive | Centralised | 13 | 12 | Absent | Absent | Absent | Pass |
| 66 | Rangasamy | 51 | Cooly | yes | 30 | Whisky | 45 | positive | positive | Centralised | 33 | 33 | Present | Present | Present | Refer |
| 67 | Velayutham | 52 | Cooly | yes | 22 | Whisky | 35 | positive | positive | Centralised | 18 | 20 | Present | Present | Present | Refer |

PTAR - Pure Tone Audiometry Notch Right Ear
PTAL - Pure Tone Audiometry Notch Left Ear
HTR - Hearing Threshold Right Ear, HTL Hearing Threshold Left Ear
RTR - Rinne's Test Right Ear, RTL - Rinne's Test Left Ear
PTA - Pure Tone Audiometry Notch,
Unit -Unit of Alcohol per week